THE JOURNAL OF PERIODONTOLOGY

April
1950

Published by
THE AMERICAN ACADEMY OF PERIODONTOLOGY

VOLUME 21
NUMBER 2

The Journal of Periodontology
is dedicated to the memory of
Dr. Gillette Hayden
for her pioneer efforts and
selfless devotion in behalf
of periodontia and the
American Academy
of Periodontology

THE JOURNAL OF PERIODONTOLOGY

(*J. Periodont.*)

Published Quarterly—January, April, July, and October—By

THE AMERICAN ACADEMY OF PERIODONTOLOGY

Mail subscriptions to

Publication and Editorial Offices: 1121 WEST MICHIGAN ST., INDIANAPOLIS 2, INDIANA

Single copies of this number may be obtained from

Publication Office at $1.50 each

Subscription Price $6.00

All foreign subscriptions to be paid in United States money

Editor Emeritus—Grace Rogers Spalding, D.D.S.

Editor—Maynard K. Hine, D.D.S.

1121 West Michigan St., Indianapolis 2, Indiana

ASSOCIATE EDITORS

Harold J. Leonard, D.D.S., 745 Fifth Ave., New York 22, N. Y.

Arthur H. Merritt, D.D.S., 580 Fifth Ave., New York 19, N. Y.

Celia Rich, D.D.S., 1112 Bennie-Dillon Bldg., Nashville 3, Tennessee

CHAIRMAN, SUBSCRIPTION COMMITTEE

E. Romle Romine, D.D.S., 1710 David Broderick Tower, Detroit 26, Michigan

AMERICAN ACADEMY OF PERIODONTOLOGY

PRESIDENT	SECRETARY	TREASURER
Dickson G. Bell, D.D.S.	Clarke E. Chamberlain, D.D.S.	Raymond E. Johnson, D.D.S.
450 Sutter Street	115 North St.	824 Lowry Medical Arts Bldg.
San Francisco, California	Peoria 5, Illinois	St. Paul 2, Minnesota

THE JOURNAL OF PERIODONTOLOGY

April
1950

Published by
THE AMERICAN ACADEMY OF PERIODONTOLOGY
VOLUME 21
NUMBER 2

CONTENTS

Nutrition and Diet in Periodontic Practice	SAMUEL CHARLES MILLER HARRY ROTH GEORGE J. WITKIN	59
Antibacterial Agents in the Treatment of Periodontal Disease	T. C. DANIELS	66
Announcement of Annual Seminar		69
The Periodontal Lesion (Part Two)	DICKSON G. BELL ROBERT W. RULE, JR. BENJAMIN DIENSTEIN JAMES NUCKOLLS	70
The Bottom of the Pathologic Crevice	A. B. RIFFLE	78
The Gingival Structures in Diabetes Mellitus	HAROLD G. RAY BALINT ORBAN	85
A Case of Acute Gingivitis Apparently Caused by Dicumarol	JESSE L. WILLIAMS	95
Editorials		96
Biomechanics of Periodontal Disease	ERNEST R. GRANGER	98
The Problem of Function in Periodontal Treatment	WILLIAM BRANSTAD	105
In Our Opinion (More About the Occlusal Factor in Periodontal Disease)	HAROLD J. LEONARD AND NINE CONSULTANTS	109
Announcements		121 - 124

Publication in the JOURNAL OF PERIODONTOLOGY *is not to be understood as endorsement by the Journal or the* AMERICAN ACADEMY OF PERIODONTOLOGY *of opinions expressed in any article, abstract or extract. The object of publishing the various views is to present them without prejudice, to the reader for his own evaluation.*

THE JOURNAL OF PERIODONTOLOGY is published to serve as the professional journal of periodontia; to keep dentists in touch with research being carried on in this field and related sciences; to stimulate greater interest in the supporting tissues of the teeth and their relation to oral and general health.

The Journal is the official organ of the American Academy of Periodontology and will contain, therefore, those papers of scientific and clinical interest which are presented at the annual meetings of that society.

Tenth Postgraduate Seminar of
American Academy of Periodontology
May 25, 26, 27, 1950—Ann Arbor, Michigan
See announcement page 69

Thirty-fourth Annual Meeting
THE AMERICAN ACADEMY OF PERIODONTOLOGY
October 26, 27, and 28, 1950
Atlantic City, N. J.

JAMES ALEXANDER SINCLAIR, D.D.S., F.A.A.P.

Early in its history the American Academy of Periodontology took steps toward the formulation of a plan for paying tribute to those whether members of the dental profession or not, who had made significant contributions to the progress of dentistry, especially in the field of periodontology.

After prolonged study by a committee appointed for that purpose, it was decided to create a Fellowship making the recipient of the honor a Fellow of the American Academy of Periodontology. In setting up the rules governing its bestowal it was emphasized that it should be given to those only who had rendered a service of distinct and recognized value to the dental profession—a rule that has been scrupulously observed throughout the intervening years, thus making the conferring of a Fellowship a distinct honor. Since the appointment of the Fellowship Committee in 1931, it has been awarded to but eighteen, of whom seven are deceased.

At the annual meeting of the American Academy of Periodontology in San Francisco in October 1949, Fellowship was awarded to James Alexander Sinclair, D.D.S., of Asheville, N. C., in recognition of his many contributions to the advancement of the dental profession.

Dr. Sinclair, a graduate of the Baltimore College of Dental Surgery, holds membership in many organizations, including Psi Omega and Omicron Kappa Upsilon dental fraternities; American Association for the Study of Neoplastic Diseases; International Association for Dental Research; North Carolina State Dental Association (President 1914-1915); American Association for the Advancement of Oral Diagnosis (President 1935); and The Southern Academy of Periodontology (President). He was a consultant in Oral Surgery, U. S. Veterans Hospital, Oteen, N. C. Dr. Sinclair is also the author of many articles on vitamin deficiencies, blood dyscrasia, anti-bioties, etc.

The American Academy of Periodontology in bestowing upon Dr. Sinclair this deserved honor, takes pleasure in wishing him Godspeed in his continued efforts to advance the art and science of dentistry.

A. H. M.

THE JOURNAL OF PERIODONTOLOGY

APRIL, NINETEEN HUNDRED FIFTY

Nutrition and Diet in Periodontic Practice

By SAMUEL CHARLES MILLER, D.D.S., F.A.C.D., F.A.D.M.,* HARRY ROTH, B.S., D.D.S.,** GEORGE J. WITKIN, A.B., D.D.S.**

INTRODUCTION—NUTRITION IN MEDICINE AND DENTISTRY

DENTISTRY may well be proud of its contribution to the science of nutrition in which it took the lead so many years ago. In a comparative study of the medical and dental literature in the years 1931-1933, it was found that 51 articles on nutrition were published in dental journals, while during the same period only seven were published in medical journals. It was not until 1938 that the proportion became equalized at 8.7% in each group.

As late as 1943, Russell M. Wilder in a paper on "The Teaching of Nutrition in Schools of Medicine" said, "The apathy of most doctors regarding dietetics is astonishing. It is due, in part, to absorption in other fields of therapeutics. However, in large part, it also comes from lack of prior training in nutrition and thus from lack of knowledge of the subject. The simplest mathematical calculation seems to be beyond the grasp of many doctors. Counting calories and balancing intake and output of nitrogen or other nutrients present insurmountable mental hazards. Yet much of modern medicine requires appreciation of the need for precision in its application. The haphazard therapy of the past is gradually giving place in medicine to bioscientific engineering."[1]

Before discussing methods of application, one is confronted with the question, "Does the dentist have the right to prescribe diet?" A strong affirmative stand must be taken. The dentist must be capable of treating all diseases of the mouth and this includes oral manifestations of nutritional deficiencies. He should be able to prescribe the type and composition of the diet with an eye to its direct local effect on the teeth, viz., detergency, alkaline-acid ratio and consistency. He should know the means of production as well as elimination of these oral effects. He should not undertake to treat conditions which are extraneous to his province such as diabetes, ulcerative colitis or anemia. However, he must be able to cooperate intelligently with the physician so that the diet which the physician advises will not be inimical to the health of the periodontal oral tissue. See fig. 1. One or two examples are appropriate here: The soft pulpy diet some

*Professor of Periodontia and Chairman of Periodontia Department, New York University College of Dentistry.

**Assistant Professors of Periodontia, New York University College of Dentistry.

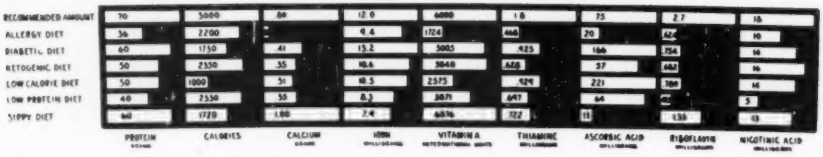


Fig. 1. Amounts of nutrients supplied by certain therapeutic diets compared with amounts recommended for an adequate diet.

physicians advise for gastric or duodenal ulcers is far from beneficial to the teeth and periodontal tissues and interferes with the dentists' control of oral disease. With a broader approach mutual benefit can be obtained by the choice of foods that will not create such hazards and will still be of therapeutic value. Another example is the prescription by some orthopedists and endocrinologists of powerful forms of vitamin D, e.g., ertron, dihydrotachysterol which raise the serum calcium by withdrawing the minerals from the bony tissues. The alveolar process being most susceptible to change is demineralized rapidly with resultant loosening of the teeth and exaggeration of periodontal disease.

PLACE OF NUTRITION IN THE ETIOLOGY OF PERIODONTAL DISEASE

The entire approach to the nutrition course in a large eastern dental school was recently changed because of the challenging statement of a keen dental student: "If we had only studied Mr. M's diet when he first came to the periodontia clinic, we might have treated him more successfully."[2] Several conflicting opinions exist concerning the etiology of periodontal disease. There are those who say that the cause is unknown. We share the opinion with many teachers that the cause is known but is never single, involving usually a combination of dysfunctional, irritational, and systemic factors in varying proportions. Diet may affect the periodontal tissues both by its direct oral action and through its systemic effect. See fig. 2.

In the *dysfunctional factors* such items as excessive stress on the teeth, too powerful masticatory musculature, premature wear and indolent mastication are all related to the consistency and type of food as it enters the oral cavity.

In the *irrational factors* must be included the nondetergent diet, food impaction and the effects of the retention of foods such as protein putrefaction and carbohydrate degradation. A recent study by Butcher indicates

*Etiology of Periodontal Disease**

(S. C. Miller Classification)

Dysfunctional

1. *Overfunction*
2. *Underfunction*
3. Abnormal habits

Irritational

1. Abnormal anatomy
2. Deposits on teeth
3. *Nondetergent diet*
4. *Food impaction*
5. Mechanical irritants
6. *Chemical irritants*
7. Atmospheric irritants (mouthbreathing)
8. Improper toothbrushing
9. Improper orthodontic procedure

Systemic

1. *Faulty nutrition*
   a. Acid-base disbalance
   b. Vitamin deficiency
   c. Mineral deficiency
2. *Debilitating diseases*
3. *Blood dyscrasias*
4. *Endocrine dysfunction*
5. *Pregnancy*
6. *Allergies* and drug idiosyncrasies
7. Metallic poisoning
8. Roentgen, radium and atomic radiation
9. *Psychosomatic factors*

Fig. 2. Etiology of Periodontal Disease. Those factors which are in italics are related to nutrition and diet.

*From S. C. Miller, Textbook of Periodontia (ed. 3), Philadelphia, The Blakiston Co., 1950.

the loss of protective cornification of the gingival and oral tissues when oily substances are permitted to remain on the mucosal surfaces.

Systemic factors should include in addition to all the obvious effects of nutrition and malnutrition on the endocrines, nervous system, vasomotor system and blood forming organs, the important periodontal and oral manifestations of acid-base disbalance, vitamin deficiency, protein deficiency, water depletion and mineral deficiencies.

DETECTION AND CORRECTION OF NUTRITIONAL DEFICIENCIES

The methods of detection and correction of all these factors must be known if the periodontist is to successfully cope with the many difficult and trying cases that confront him. It obviously is most important to look at the patient as a whole being rather than as a series of loosely connected entities to be separately repaired. Thus a primary responsibility of our profession is to regard the optimal functioning of the entire body. In order to discharge this responsibility successfully, our attention should be directed to the nutritional well being of the patient.

The direct effect of dietary deficiencies may produce any or all of the following disorders:

1. Lowered resistance of oral tissues to the influence of local irritants and infection.
2. Lowered pain threshold.
3. Prolonged clotting time.
4. Prolonged bleeding time.
5. Delayed healing.
6. Inflammatory reactions in the temporomandibular joints.
7. Specific effects on the periodontal tissues.
8. Specific effects on the teeth, e.g.—poor structure in formation, malocclusion, or insufficient or excessive wear.

While diet may be described broadly as the usual food and drink taken daily by an individual, nutrition concerns the utilization of the food by the body. This utilization is affected by the types of food, absorption, emotional factors, etc.

The diet may vary from one which will keep the person alive (adequate diet) to one that is abundant, diversified, well balanced and which will prolong life with maximum health (optimum diet). This latter diet will supply the body with the required food elements for growth and repair of tissues and will provide an optimal amount of heat and energy.

Many anthropologists believe that the modern soft, sophisticated and nonstimulating diets tend to produce dental and oral pathologic conditions in civilized people. How do some of our present day food fads based on non-scientific "evidence" compare with those of some of our contemporary primitives? The Bemba tribe of northern Rhodesia eat a stew consisting of meath, fish, caterpillars, locusts, ants, vegetables, mushrooms, and so forth. Scientifically this diet is better balanced than that of some of our modern faddists who subsist solely on one type of food.

Modern diets may produce nutritional deficiencies primarily by inadequate food intake or secondarily in association with other diseases. Whether primary or secondary, there exist several groups of patients who are more likely to have these deficiencies. These may be classified as (modified from Jolliffe):[3]

1. Indigent and low income group.
2. Persons who have erroneous dietary habits and food idiosyncrasies, i.e., excessive use of sugar, candy, pastry, soft drinks, etc. The more varied the diet, the less likely it is to be inadequate.
3. Alcohol addicts.
4. Patients with diseases altering the nutritional requirements.
   a. Hyperthyroidism.
   b. Fevers, especially of long duration.
   c. Gastro-intestinal disturbances, e.g., colitis, achlorhydria, vagotonic spasms.
   d. Pregnancy.
   e. Rapid growth.
   f. Increased excretion, e.g., diarrhea, polyuria, lactation.
   g. Excessive ingestion of alcohol, refined starches or mineral oil.
   h. Nervous states.
5. Food faddists.
6. Those who are edentulous or are wearing inefficient dentures.

PERIODONTIA DEPARTMENT
NEW YORK UNIVERSITY
COLLEGE OF DENTISTRY
209 EAST 23rd STREET
NEW YORK CITY

FOOD DIARY

Write down everything you eat.

DATE	BREAKFAST	LUNCH	DINNER	BETWEEN MEALS List Time of Day as well as Food Taken
10-31-48 Sunday	1 tomato, lettuce 1 small whitefish 2 eggs with milk 1 glass (8 oz.) milk 1 large slice chocolate cake	1 western sandwich, 1 frankfurter with mustard & relish 1 cup coffee, 2 lumps sugar	Steak, 2 potatoes, green peas, string beans, 5 slices white bread 1 glass soda	2 glasses milk
11-1-48 Monday	1 glass (8 oz.) grapefruit juice 1 cup coffee. Roll & butter, 1 glass milk	beef, noodles 2 sweet rolls coca cola	5 salmon croquets, spaghetti, string beans 5 slices rye bread	2 glasses milk 1 ice cream soda.
11-2-48 Tuesday	1 glass milk 1 glass grapefruit juice. Roll and butter. 1 cup coffee, 2 teaspoons sugar	Chopped egg sandwich, 1 lettuce, tomato chocolate malted	2 lamb chops, potatoes, 6 slices rye bread 1 glass soda	grapes. 2 glasses milk
11-3-48 Wednesday	1 glass grapefruit juice, roll & butter 1 cup coffee, 2 teaspoons sugar	Veal cutlet potatoes, carrots peas 4 slices rye bread	Steak, potatoes 1 glass soda Fruit cocktail 5 slices rye bread.	2 glasses milk
11-4-48 Thursday	1 glass orange juice. 2 scrambled eggs 1 glass milk	—	Tuna & tomato sandwich chocolate malted. plain cake	½ lb. cashew nuts 2 glasses milk
11-5-48 Friday	Roll & butter 1 cup coffee with milk sugar 1 glass grapefruit juice	Western omelet, 2 rolls potatoes, string beans	7½ ounces spaghetti, lettuce & tomato	1 glass milk
11-6-48 Saturday	1 cup coffee with milk & sugar Roll & butter		Tuna & tomato sandwich chocolate malted 1 slice plain cake	2 glasses milk.

This chart modified by Lillian Lee D.H. from the U.C. Juvenile Food Diary.

Patient's Name Ronald Zimmerman.

11-16-48

Fig. 3. Patient's weekly diet record.

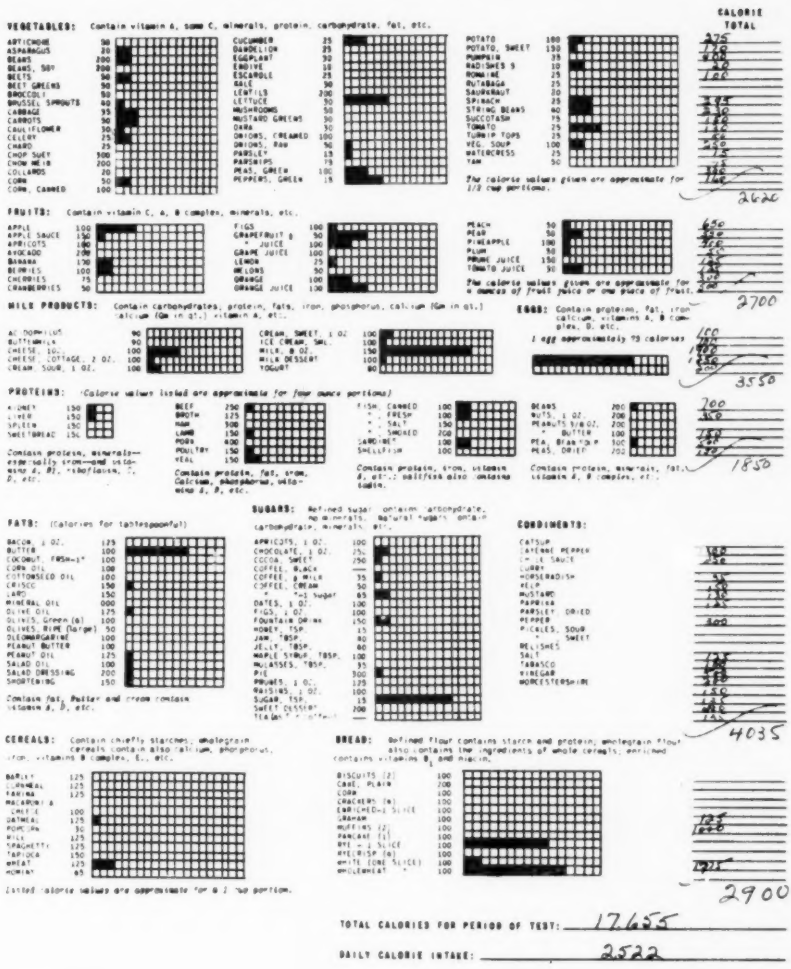

A SIMPLIFIED GUIDE FOR EVALUATING DIET HISTORIES
Periodontia Dept., B.Y.U. College of Dentistry
VEGETABLES: Contain vitamin A, some C, minerals, protein, carbohydrate, fat, etc.
FRUITS: Contain vitamin C, A, B complex, minerals, etc.
MILD PRODUCTS: Contain carbohydrates, protein, fats, iron, phosphorus, calcium (Gm in qt.) calcium (Gm in qt.) vitamin A, etc.
EGGS: Contain protein, fat, iron, calcium, vitamins A, B complex, D, etc.
1 egg approximately 75 calories
PROTEINS: (Calorie values listed are approximate for four ounce portions)
Contain protein, minerals—especially iron—and vitamins A, B1, riboflavin, C, D, etc.
Contain protein, fat, iron, Calcium, phosphorus, vitamins A, B, etc.
Contain protein, iron, vitamin A, etc.; shellfish also contains iodine.
Contain protein, minerals, fat, vitamin A, B complex, etc.
FATS: (Calories for tablespoonful)
SUGARS: Refined sugar contains carbohydrate, no minerals. Natural sugars contain carbohydrate, minerals, etc.
CONDIMENTS:
Contain fat. Butter and cream contain vitamin A, D, etc.
CEREALS: Contain chiefly starches, wholegrain cereals contain also calcium, phosphorus, iron, vitamins B complex, E., etc.
BREAD: Refined flour contains starch and protein, wholegrain flour also contains the ingredients of whole cereals; enriched contains vitamins B1 and niacin.
Listed calorie values are approximate for 0.1 cup portion.
The calorie values given are approximate for 1/2 cup portions.
The calorie values given are approximate for 4 ounces of fruit juice or one piece of fruit.
CALORIE TOTAL
2620
2700
3550
1850
4035
2900
TOTAL CALORIES FOR PERIOD OF TEST: 17655
DAILY CALORIE INTAKE: 2522
RECOMMENDATIONS:
VEGETABLES: Adequate
FRUITS: Adequate
MILK: Adequate
CHEESE: Adequate
EGGS: Adequate
FATS: Adequate; Pleasings - Adequate
SUGARS: Adequate
CEREALS: Whole grain Cereals
BREAD: Adequate
VITAMIN OR MINERAL SUPPLEMENTS:
NAME: Fred Abram
DATE: 11-16-48

Fig. 4. Simplified guide for diet analysis.

NEW YORK UNIVERSITY
COLLEGE OF DENTISTRY
DEPARTMENT OF PERIODONTIA

SPECIFIC DIETARY ADVICE for:

M..... Date.....

Signed.....

(See other side for elements of a complete diet.)

NEW YORK UNIVERSITY
COLLEGE OF DENTISTRY

Department of Periodontia

The proper diet for a *normal* individual *should include* the following:

1. *Fruits*—oranges, grapefruit, raspberries, apples, pineapples, prunes, bananas, etc.
2. *Vegetables*—all in abundance, especially carrots, lettuce, celery, cabbage, cauliflower, string beans, green peas, beets, asparagus, spinach, tomatoes, etc. Some raw vegetables every day. Never boil vegetables—steam them.
3. *Two to four glasses of milk a day;* cream, dairy products, etc. (Take milk at meal times.) Do not use chocolate flavors or jam in milk.
4. *Mild cheeses*—cottage, cream, etc.
5. *Butter*—three teaspoonfuls daily.
6. *Cod Liver Oil*—one teaspoonful a day. Sept. 15th to June 15th.
7. *Soups*—creamed, chowder, pureed, etc.
8. *Meats*—organ meats (liver, brains, sweetbreads) at least once a week. Muscle meats (steaks, beef, chops, etc.), three times a week.
9. *Fish*—fresh cod, halibut, bass, or lake fish, once or twice a week.
10. *Eggs*—one or two daily, not fried.
11. *Bread*—whole wheat, rye or pumpernickel—sparingly.
12. *Cereals*—whole wheat—sparingly.
13. *Sweets*—natural, dates, raisins, figs, brown sugar, honey.
14. *Water*—at least three to four glasses a day.

(See other side for specific recommendations)

Fig. 5. Diet prescription card.

Our approach to the study of a patient's diet gives us the opportunity to adequately evaluate their food intake. One must realize that a patient's diet history record is only an approximate estimation. A scientifically exact measurement of a diet would be impractical other than for research purposes.

In the Periodontia Department of New York University College of Dentistry, we have tried many charts and systems for recording a patient's diet. The following method is the one finally decided upon that will meet needs of the periodontist for history taking, analysis and specific recommendations.

The diet history is a record of a patient's weekly diet as well as a history of the mouth care and personal habits. See fig. 3.

The front of the chart contains squares in which the patient enters his entire food intake for breakfast, lunch, dinner, and everything eaten between meals. The reverse side contains questions concerning food habits, past and present medical history and mouth care.

When the patient is given the food diary, several points must be stressed in order to insure accuracy. First, it is most important that the normal diet be followed for the test period and all foods eaten be listed. Each meal is entered as soon after the meal as possible and all foods are considered average portions unless specifically stated. As an example, the patient may enter 2½ slices white toast with butter or 1 cup coffee with cream (1 oz.) and 1 teaspoonful of sugar. Care must be taken to be certain that normal

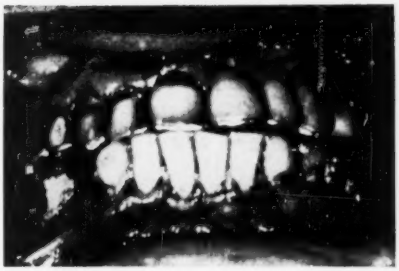


Fig. 6. Desquamative gingivitis associated with menopause (age 55). Unsuccessful periodontal treatment for three years. Diet very high in refined carbohydrates. Fig. 6a. Before nutritional therapy. Fig. 6b. One year later. Substitution of high vitamin and protein diet. Supplemented multiple vitamin and dessicated liver therapy. No other treatment given during this period.

portions are mutually understood, for while a 4 oz. steak may be a normal for one person, another will consider an 8 oz. steak normal.

The answers to the questions on the reverse side of the chart are made at the patient's leisure by a check or single word.

From this history one proceeds to the diet evaluation chart which is a simplified guide[4] for analyzing a patient's diet history. See fig. 4. The use of this chart makes possible a quick and accurate analysis of the types of food eaten and their caloric values.

The chart itself has individual foods grouped together under headings as vegetables, fruits, milk products, proteins, etc. For each group of foods, the size portion is stated (½ cup, 4 ounces or 1 piece of fruit, etc.) and the number of calories for the individual food of that size portion is given. Following the caloric value are a series of ruled boxes to be used in the actual evaluation. Thus for example we may find that under the heading of "proteins," (the size portion is 4 oz.) beef has a caloric value of 250.

Each food listed in the diet history by the patient is transferred to the evaluation chart by blackening a ruled square next to the individual food. After all the history is transferred, there will be a scattered series of blackened squares which will show at a glance the relative distribution of the quantity of foods within each group.

The right side of the evaluation chart has a column for the total caloric value of the test period. From this the daily intake is computed.

With all the organized information at hand, the final dietary recommendations for the patient can be easily and accurately made. On the bottom of the chart spaces are provided for the recommendations which are then copied on a diet prescription card which is given to the patient.

This diet prescription card (see fig. 5) supplies the means for giving the patient the elements of a complete diet as well as specific dietary advice for that particular patient.

One side of the card summarizes the proper diet for an average individual. The reverse side is completed with the recommendations derived from the evaluation of the patient's diet history. This card is given to the patient.

The foregoing procedure has been used for the past ten years with favorable results to both patient and periodontist. The patient is impressed with the comprehensive dietary analysis. Cooperation in all phases of periodontal treatment is enhanced.

The periodontist is aware that treatment must be given to the whole patient. He must be concerned not only with the removal of local effects of a periodontal syndrome, but with all vitiating factors, whether they are local or systemic.

BIBLIOGRAPHY

1. Wilder, R. M., Teaching of nutrition in schools of medicine, J. A. Am. M. Coll. **18**:145-157, May 1943.
2. Committee of Professional Education, Am. Dietet. A., Nutrition education in dental schools, Jour. Den. Educ. **5**:15, Oct. 1940.
3. Miller, S. C., Textbook of Periodontia (ed. 2), Philadelphia, The Blakiston Co., 1943, p. 480.
4. Blass, J. Lewis, Simplified guide for evaluating diet histories, J. D. Med., **2**:43-46, April, 1947.
5. Bowes, A. De., Teaching of nutrition to medical and dental students, J. Am. Dietet. A. **15**:776-7, Nov. 1939.
6. Butcher, Earl O., A.M., Ph.D., Professor of Anatomy, New York University College of Dentistry, Personal communication.
7. Klatsky, Meyer, Studies in the dietaries of contemporary primitive peoples, J. Am. Dietet. A. **36**:385-391, April-May 1948.
8. Miller, S. C., Oral Diagnosis and Treatment (ed. 2), Philadelphia, The Blakiston Co., 1946.
9. Newburgh, L. H. and Light, A. M., Teaching of nutrition to medical students, J. Am. Dietet. A. **21**:209-210, April 1945.
10. White, R. L., Dietitian as teacher of dental students, J. Am. Diet. A. **18**:375-378, June 1942.
11. Whitehead, F. E., Research in nutrition education, J. Am. Dietet. A., **23**:310-317, April 1947.

Antibacterial Agents in the Treatment of Periodontal Disease

T. C. DANIELS, B.S., Ph.D., *San Francisco, California*

IT HAS long been the goal of chemotherapy to find specific agents for the control of localized or systemic infections. Paul Ehrlich who was first to develop the concept of chemotherapy stated the ideal therapeutic agents consist of "magic bullets which strike only those objects (that is, the pathogens) for whose destruction they have been produced." While the chemotherapeutic agents now in use usually fall somewhat short of Ehrlich's *beau ideal*, many of them approach his dream and without question represent great advances over the compounds formerly available. As a result of this development many medical and dental problems dealing with therapeutics have been greatly simplified or entirely resolved. This new era of chemotherapy was inaugurated with the development of the sulfonamides and was accelerated by introduction of the antibiotics.*

This paper is concerned with some of the new agents that appear to be best suited for the treatment of periodontal disease. Most of the compounds to be considered fall into the general class of agents known as antiinfectives. At best, these must be regarded as aids to supplement accepted conservative surgical procedures in the handling of periodontal lesions. Although periodontal lesions are normally treated topically, it should be kept in mind that they may serve as focal points for serious systemic infections. This hazard may be much greater than is generally realized. The lesions are subjected to constant manipulation from the chewing of

University of California, College of Pharmacy.

Presented at the Thirty-third Annual Meeting of The American Academy of Periodontology, San Francisco, California, Oct. 1949.

*It is also of interest to note that comparable advances have been made in the development of other therapeutic agents not dealing with infections.

food and this may lead to the introduction of localized infections into the blood stream. When this occurs and systemic infections are involved, localized treatment must be supplemented with systemic therapy.

Periodontal lesions

The normal aging epithelium, although undergoing continuous desquamation, presents a comparatively uniformly smooth surface. On the other hand, the cell erosion associated with a periodontal lesions leads to the formation of cellular and tissue fragments both living and dead. This necrotic process involves both the epithelial and connective tissues in which there are formed pockets, fissures, and cellular debris in varying stages of degradation. The capillaries in the soft tissue tend to become dilated. This tends to decrease capillary blood flow and to lead to localized anoxia. Likewise, the soft tissues undergo changes in permeability leading to swelling. These overall changes in the tissues complicate the problem with respect to the accessibility of the surfaces involved for debridement and treatment.

Surface Active Agents

Solutions having a low surface tension are required to penetrate the tissues and bring the active-infective agent into immediate contact with the otherwise inaccessible surfaces. By making judicious use of surface active agents, the dentist has a better chance of successfully treating periodontal lesions and thereby minimizing the need for radical surgical procedures. Fortunately, there are available surface active agents (wetting agents) that will not lower the activity of the more commonly used anti-infective agents. Indeed, in some cases the activity may be enhanced. However, it should be kept in mind that the surface active agent cannot be selected at random without the possibility of lowering or even completely inactivating the antibacterial compound selected for use. As an example, cationic (basic) antibacterial agents (e.g. 9-aminoacridine, zephiran, etc.) are normally incompatible with the anionic (acid) type (e.g. soaps, sulfonated alcohols, etc.) wetting agents. This antagonism appears to be due to the formation of slightly dissociated insoluble salts from the high molecular weight anions (acids) and cations (bases). On the other hand, cationic antibacterial agents are normally compatible with cationic and neutral surface active agents. Likewise, anionic antibacterial agents can ordinarily be used with anionic or neutral surface active agents.

ANTIBACTERIAL AGENTS AS ADJUNCTS IN THE TREATMENT OF PERIODONTAL LESIONS

Peroxides

The peroxides have been employed for a number of years in the treatment of periodontal lesions. As a group they offer four important properties consisting of *antibacterial action, mechanical cleansing, oxygenation of tissues* and *deodorization*. The antibacterial activity in so far as is known is proportional to the oxidizing power, which in turn is related to the concentration of the peroxide. The mechanical cleansing is due to the release of nascent oxygen in tissue pits and fissures. As the oxygen gas is released, it carries with it to the lesion surface loose cellular fragments from the necrotic process. The tissues in a periodontal lesion are usually under low oxygen tension due at least in part to the dilation of the blood capillaries and laking of the capillary blood resulting in tissue anoxia.

The peroxides supply nascent oxygen directly to the tissues and convert methemoglobin to hemoglobin thereby restoring a normal transitory oxygen tension. It should be kept in mind that this action is of comparatively short duration. The peroxides most commonly used are hydrogen peroxide (3-30 per cent) and urea hydrogen peroxide (35 per cent). U.S.P. hydrogen peroxide (3 per cent) and Superoxol (30 per cent) are both acidic in character and are somewhat unstable with surface active agents. Urea hydrogen peroxide may be used in anhydrous propylene glycol or glycerin or may be employed as a stabilized powder in combination with urea and a wetting agent. A

fresh solution of the stabilized powder in water can be prepared as required for use. This solution has low surface tension and good wetting properties which bring the peroxide in intimate contact with all accessible surfaces of the lesion. The urea in urea hydrogen peroxide appears to have advantages of its own in that it tends to solubilize proteins and dead cell fragments without harm to normal cells. In this way it serves as an agent for chemical debridement and increases the accessibility of the tissue surfaces for the antibacterial agent selected for use. In view of the above properties of the peroxides, it would appear desirable for them to be employed just prior to the use of other antibacterials.

Sulfonamides

The sulfonamides at one time enjoyed considerable popularity in the treatment of periodontal lesions. However, more effective agents are available at the present time and there appears to be little to support their continued use.

Antibiotics

During the last decade many antibiotics have been developed for commercial distribution. A number of them offer a bacterial spectrum and general physical properties that make them well suited for experimental use in the treatment of periodontal lesions.[1] The following antibiotics appear to be appropriate for this purpose.

Penicillin

Penicillin was the first antibiotic to be commercially distributed and it has received wide and successful use in dental practice for the treatment of periodontal disease. This antibiotic has at least two important disadvantages. First, drug sensitivity is reported in approximately 10 per cent of the cases in which it is topically applied. Individuals who have had a history of fungus infections such as athlete's foot are reported to show a much greater tendency to sensitivity. The second disadvantage of penicillin is its property of prolonging the clotting time of blood[2] which may possibly lead to complications in its use both for extractions and surgical treatment of periodontal lesions. Penicillin also has the disadvantage of occasionally inducing development of strains of bacteria that are penicillin resistant. Except for these disadvantages, penicillin is perhaps the drug of choice for treatment of infections involving penicillin sensitive Gram positive organisms: Penicillin has little or no action against most Gram negative organisms. The drug is normally used in the form of lozenges or as an ointment. Preferably, local application of penicillin should be augmented by systemic treatment.

Tyrothricin

Tyrothricin has approximately the same bacterial spectrum as penicillin and is a useful substitute for penicillin in penicillin sensitive patients. Like penicillin, it has little or no action against most Gram negative organisms. The drug is available only as a lozenge and ointment and is not suitable for parenteral use.

Bacitracin

Bacitracin has a similar bacterial spectrum to penicillin but appears to have less tendency to develop drug resistant strains of microorganisms. This antibiotic is presently available for use as an ointment.

Polymyxin A

This antibiotic is not at present commercially available but can be obtained in small quantities for experimental use. Some of the polymyxins studied are quite toxic when introduced into the blood stream but this disadvantage is largely overcome in polymyxin A. Its principal advantage is its activity against Gram negative organisms for which many of the antibiotics show little or no activity.

Aureomycin and Chloromycetin

Both aureomycin and chloromycetin (Chloramphenicol) are active against Gram negative and Gram positive organisms. Because of this wide bacterial spectrum, they are of considerable interest for experimental

use in the treatment of periodontal lesions. Chloromycetin may be incorporated in an ointment for experimental use and aureomycin is available as an ointment and in lozenge form.

Miscellaneous Agents

9-Aminoacridine shows good activity against both Gram negative and Gram positive organisms. It may be used in specially formulated preparations as a solution of 0.05 to 0.1 per cent concentration or as an ointment in the same concentration. This compound is a cationic (basic) antibacterial agent and is incompatible with a number of high molecular weight anions (acids). Only those products that have been thoroughly tested are suitable for use.

Nitrofurazone

The semicarbazone of 5-nitro-2 furfuraldehyde is distributed under the name of Furacin. This compound is active against a number of Gram negative and Gram positive organisms and is suitable for experimental use in the treatment of periodontal lesions. The compound is available as an ointment and in solution, each containing 0.2% of the nitrofurazone.

SUMMARY

The periodontal lesion presents special problems for effective topical treatment. The use of surface active agents in combination with active antibacterial agents is briefly discussed and a number of the compounds currently available for use by the periodontologist are considered. Although most of these compounds are highly active antibacterial agents they should be regarded only as adjuncts in the overall treatment of periodontal disease.

The author is indebted to Dr. James Nuckolls, Professor of Operative Dentistry and Dr. Harry Frisbie, Associate Professor of Dentistry of the College of Dentistry and to Dr. Robertson Pratt, Associate Professor of Pharmacognosy and Plant Physiology, College of Pharmacy for their interest and suggestions.

REFERENCES

1. Pratt, Robertson, and Dufrency, Jean: Antibiotics. J. B. Lippincott Co., Philadelphia (1949).

2. Hasselbroch, W. B., and Cateno, Carlos. Science. **102**:38-40 (1945).

Annual Seminar to Be Held May 25, 26, 27, 1950

The Annual Seminar of the Academy of Periodontology which is sponsored by the Kellogg Foundation will be held at Ann Arbor, Michigan, on Thursday, Friday and Saturday, May 25, 26 and 27, 1950.

The program will consist of discussions in psycho-somatics, endocrinology, occlusal force related to periodontal disease, biochemistry, histochemistry and blood dyscrasia.

The Seminarians will be quartered at the Michigan Union. Rooms have been reserved at the Michigan Union for a number of members and applications should be made directly to the Michigan Union for assignment to these rooms.

The registration fee is $25.00 and application for attendance should be made directly to Dr. Paul H. Jeserich, c/o Kellogg Foundation, University of Michigan, Ann Arbor, Michigan. Members of the Academy will have priority for registration. A limited number of guests may be accepted if Academy members do not fill the registration of about thirty. A non-member guest should be vouched for by an Academy member.

Dr. Robert G. Kesel is Chairman of the Post Graduate Study Course Committee.

The Periodontal Lesion

II. A Roentgenographic Evaluation of Treated Periodontal Cases
A biological approach*

DICKSON G. BELL AND ROBERT W. RULE, JR. IN COLLABORATION WITH
BENJAMIN DIENSTEIN AND JAMES NUCKOLLS

INTRODUCTION:

PERIODONTITIS is not a disease from which a patient can be considered "cured" following a course of therapy —unless that therapy be the extraction of the teeth. It is a disease which must be continually kept in abeyance, and, therefore, one which demands continued prophylactic measures. Because the periodontist is faced with a problem of maintaining a periodontium at optimal function and repair, he necessarily must adhere to sound biological principles in his treatment, utilizing the accepted studies on tissue metabolism in health and disease.

It is one thing to theorize and set up a plan of treatment under these conditions and another to put those principles to practice and evaluate them after a long period of time.

A roentgenographic evaluation of a long-term periodontic study is herewith reported. The cases have been systematically followed over various periods of time up to 25 years. In making these evaluations all of the known etiological factors—local, systemic and psychological—have been brought to bear on the analysis.

It is realized that 25 years ago treatment was based, to a great extent, on empiricism. While a certain knowledge of tissue reaction was appreciated and applied in practice in the past, it was necessary to await the advent of the biochemical sciences for interpretation of the more subtle tissue changes.

The purpose of this paper is to substantiate the theoretical concept that the development of a rational approach to therapy on the knowledge of tissue reactions can result in a functional repair and arrest of the progress of periodontosis for considerable lengths of time.

*Part I published in Jour. of Perio. 21:7 (Jan.) 1950.

REVIEW OF THE THEORETICAL CONCEPT OF THE DEVELOPMENT AND ESTABLISHMENT OF THE LOCAL PERIODONTAL LESION WITH A BRIEF REFERENCE TO SYSTEMIC INFLUENCES*

Inflammation may be defined as a complex vascular, lymphatic and tissue response to the presence of an irritant. An irritant is any physical, chemical, or biologic agent which interrupts normal cell metabolism. The periodontic lesion is initiated by an irritant, developed by the inflammatory reaction and established either by its own efforts to heal or by a therapy which does not produce an optimal repair and function.

It has been established that the local circulation in inflamed areas is markedly impaired through the occlusion by fibrin of the arterioles and lymphatic outlets. In time the area of inflammation, circumscribed by a fibrin block, is isolated from the rest of the structure and consequently develops within itself different degrees of an anaerobic metabolism. Interference with the intermediary carbohydrate metabolism in the cells results in the formation of lactic acid, and the pH of this area drops to levels in which tissue stability cannot be maintained. Bone salts within such an area are thus caused to go into solution. If the inflammation is severe, the bone matrix becomes involved

*See "The Periodontic Lesion, Part I. The Development of the Lesion and the Establishment and Treatment of the Periodontal Pocket."

and eventually lost through the action of autolytic enzymes which are activated in an acid medium.

The changes described above are essentially biochemical in nature, and the reactions are reversible with the removal of the irritant.

The systemic factors are considered as biologic irritants, for they produce their irritation by interfering with normal cell metabolism—either directly or indirectly withholding necessary cell nutrient requirements. Psychic irritants affect cell metabolism mainly by producing peripheral vascular spasm and hence limiting the optimum essential requirements of the affected area. It has been previously pointed out that the periodontium, because of its complex anatomical relationships, makes it an ideal area for reflecting subtle local and systemic disturbances which may induce an inflammatory response through an interference in cell metabolism.

DATA PRESENTED

The cases which are to be presented have been selected for reason of their "typal" difference.* The plates will show the original roentgenograms and those of recent date. The treatment procedure has been subgingival curettage, except in several areas where the gingival tissue was resected because of unfavorable anatomical relationships, where recurrent inflammation or persistent hypertrophy existed. It is readily admitted that subgingival curettage is not the only single method of local treatment, but it does grant a first approach to the periodontal lesion. In a certain number of cases it is the only treatment that is necessary; whereas in other cases it at least permits cleaning up the areas, reducing the inflammatory reaction and preparing the tissues for further surgery.

A comparative study such as this has its difficulties, for a satisfactory reproduction of two different sets of roentgenograms on the same plate—considering the advances in the field of roentgenography during the past quarter century—presents a demonstration problem which cannot be ignored. Some slight fading of the original roentgenograms has also occurred, hence the more recent pictures will appear slightly darker. The position of the teeth on the plate will be the same as though the reader were looking at the patient. All restorative work was done by the patient's dentist.

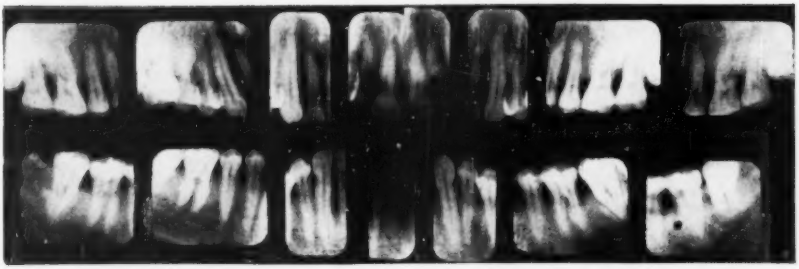
CONCLUSION

When a gingivitis or inflammatory condition exists, it indicates the presence of some irritating factor—which in many cases may be due to or complicated by systemic dysfunction. If periodontal therapy is instituted and the inflammation disappears, then the clinical conclusion must be made that the etiological factors are under control. This is proved roentgenographically by evidence of no further loss of bone structure. Histologists (with whom this question has been discussed) agree that where it can be demonstrated that no resorption of bone has occurred over a period of years, then it must be accepted that the original inflammatory process has been controlled as a result of treatment, and that no persistent chronic inflammation or infectious process has recurred in the meantime. This evidence has been shown by the roentgenographic evaluation of these treated cases. That repair of the lesion does take place was demonstrated by repair of the bone and restoration of the cortical plate.

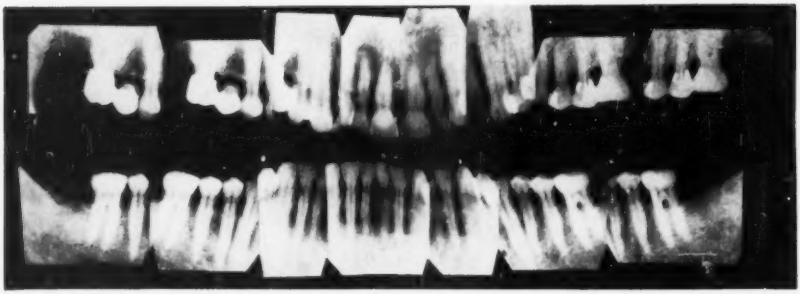
The objective of periodontal therapy, therefore, is to bring about a repair of the tissues, re-establish function and maintain this condition as long as possible. Roentgenographic and clinical records of many years show that with periodic prophylaxis, optimum diet and health, and vigilant care on the part of the patient, a functional periodontium can be maintained indefinitely.

*The Editor regrets it was necessary to reduce to six the series of twelve cases submitted by the authors.

CASE 1



INITIAL



LAST

History: 1928 Male, aged 54. Ulcer of right eye, with hemorrhagic areas, causing loss of vision—left eye slightly affected . . . patient's dentist reported no evidence of focus of infection . . . a sub-mucous resection and local treatment of eyes resulted in a temporary improvement, followed by a recurrence.

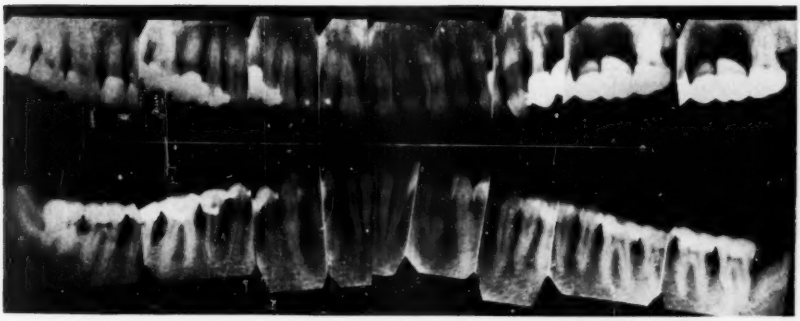
Examination: 1928 Gingivae inflamed and edematous . . . supragingival calculus on lower anteriors and upper molars . . . subgingival calculus throughout . . . generalized pocket formation . . . bifurcation involvement of all first and second molars, but no evidence of suppuration.

Treatment: 1928 Extraction of upper second and third molars, lower second molars and left third molar . . . subgingival curettage . . . instruction in mouth care . . . periodic prophylaxis.

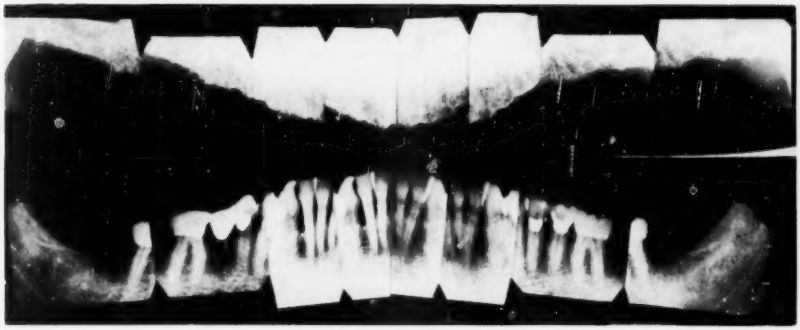
Results: 1949 Roentgenograms of 1949 show no further bone resorption—cortical plate restored in many areas . . . gingival tissues in excellent condition except for occasional marginal irritation from deposition of calculus on lingual of lower anteriors and buccal of upper molars. Eye condition immediately improved with extractions and periodontal treatment with no recurrence to date . . . marked improvement of vision.

Comment: The following factors led to a favorable periodontal prognosis: a coöperative patient, no evidence of systemic contributing factors, excellent bone structure, long roots and a moderate degree of bone destruction except in the first molar areas. Since the eye condition did not successfully respond to any treatment until the gingival tissues were treated by curettage and extraction of teeth accomplished, the source of eye pathology could have been related to the periodontal inflammation.

CASE 2



INITIAL



LAST

History: 1934 Male, age 39, height 5'9½", weight 126 lbs. Usual childhood diseases . . . called "sickly, puny child" . . . pneumonia at 17 . . . chronic constipation and fatigue . . . inferiority complex about physique; took exercises and gymnasium work but failed to develop large muscles . . . admires athletic type . . . heavy alcohol consumption 1917-1928, but moderate past six years . . . smokes at least two packages of cigarettes daily . . . slight anemia . . . BMR-13 . . . thiamine, vitamin C and D intake low, riboflavin fair, calcium fair, phosphorous high.

Examination: 1934 Gingival tissues inflamed—hemorrhagic and dark red color in many areas . . . generalized subgingival calculus—supragingival deposits in lower anterior region . . . poor mouth hygiene . . . many deep vertical pockets . . . suppuration $\frac{4 \mid 3\ 4}{5\ 2 \mid}$ —mobility of $\frac{8\ 5\ 2\ 1 \mid 3\ 4\ 7}{8\ 5\ 2 \mid\ 7}$, migration of upper central incisors, extrusion of upper left cuspid, malocclusion. Roentgenograms compared with some taken in 1932 showed rapid and extensive bone resorption in two-year period, also a lack of normal bone regeneration in upper left first bicuspid and molar area following extractions (1932).

Treatment: 1934 The physician and periodontist agreed that the first therapeutic approach should be from the systemic standpoint and employed treatment for anemia and the low thyroid condition, prescribed an adequate diet supplemented by vitamins, and a reduction in smoking to five cigarettes per day. The periodontal treatment consisted of subgingival curettage, instruction in mouth care and periodic clinical and roentgenographic examinations.

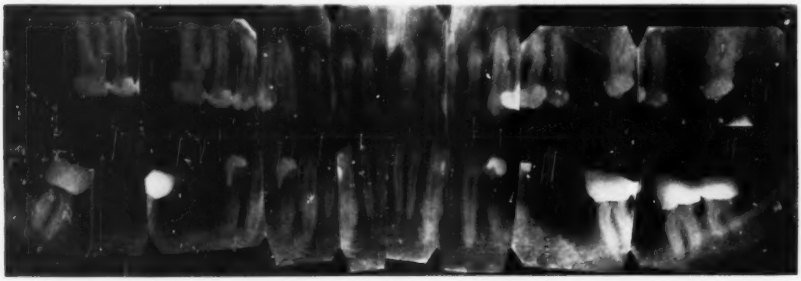
Result: 1937 Extraction of all upper teeth and $\frac{\quad|\quad}{8\ 7\ 5\ |\ 7}$.

1947 No further bone resorption.

1949 Good gingival tissue tone and color . . . no mobility or suppuration lower lateral incisor . . . decrease in pocket depth . . . no resorption of upper alveolar ridge and in past twelve years has had but one denture besides the immediate . . . has stopped smoking . . . has an occasional cocktail . . . weighs 144 lbs., and feels better than at any time in his life.

Comment: Most unfavorable prognosis from a periodontal standpoint . . . extraction of several upper teeth in 1934 was considered, but the clinical condition and roentgenographic evidence of what had occurred in the previous two years and the question of how the remaining teeth and tissues would react to restorative measures indicated this procedure was questionable. Etiologic factors were local irritation (calculus, materia alba, excessive stress), anemia, a dietary deficiency, combined with bad early background and a psychosomatic problem. In such cases, excessive stress accelerates the process of bone resorption. Much of what was accomplished was due to the coöperation of the patient.

CASE 3



INITIAL

LAST

History: 1939 Male, age 43, height 5'11", weight 153 lbs. Referred by physician . . . physical examination normal . . . unfavorable emotional background . . . irrational life pattern . . . unable to make decisions . . . 3 to 6 oz. of alcohol and 35 cigarettes daily . . . generally good dietary intake . . .hypersensitive about dental treatment.

Examination: 1939 Gingivae inflamed . . . supragingival calculus on lower incisors . . . slight subgingival calculus . . . materia alba throughout . . . generalized bone resorption—suppuration $\frac{\quad|\quad}{5\ 1\ |\ 3}$

. . . bifurcation involvement of molars . . . slight malocclusion . . . excessive stress with mobility in upper bicuspid and both anterior regions . . . $\frac{1}{|}$ loose, rotated and extruded . . . hypersensitiveness of exposed dentin on labial and buccal surfaces. Patient and physician advised of unfavorable prognosis; nevertheless periodontal treatment requested.

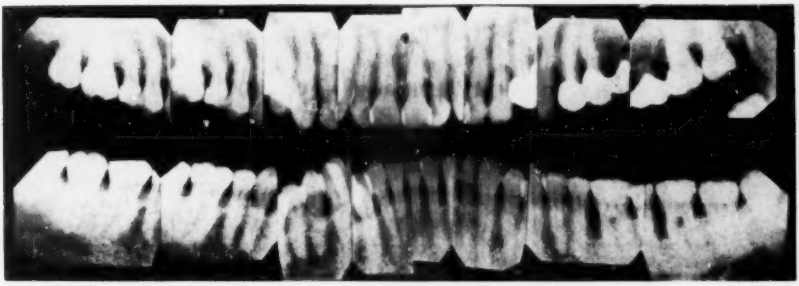
Treatment: 1939 Subgingival curettage . . . extraction $\frac{1}{|}$ . . . replacement $\frac{1 \mid 6}{}$ (had lower removable appliance) . . . excessive stress partially relieved . . . instruction in mouth care . . . increase protein intake . . . decrease alcohol and tobacco . . . supplementary vitamin B therapy.

1939-1942 Periodic prophylaxis . . . subgingival curettage . . . repeated instructions in toothbrushing and interproximal care.

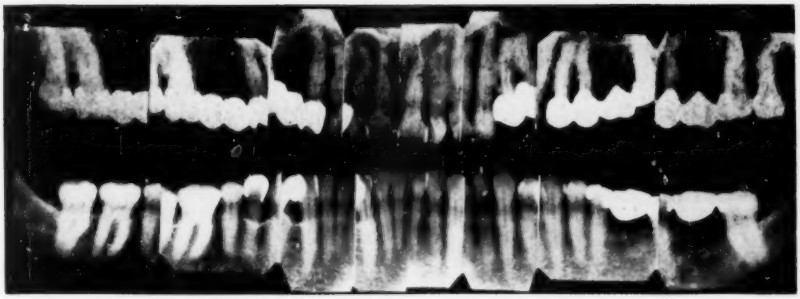
Results: 1942 Periodic recurrence of gingival inflammation . . . suppuration and increased mobility of upper teeth . . . extrusion $\frac{| 3}{}$ . . . repeated attempts at periodontal therapy under procaine unsatisfactory . . . less inflammation with no suppuration of lower gingival tissues and no increase in pocket depth or mobility of these teeth . . . roentgenographic evidence of progressive bone resorption in upper arch . . . practically no change in bone picture on the lower . . . extraction of all upper teeth advised . . . patient uncoöperative about alcohol, tobacco, mouth hygiene, dietary and vitamin recommendations . . . patient procrastinated about upper extractions . . . prophylaxis of lower teeth continued until subsequent roentgenograms showed bone resorption developing in this arch.

Comment: Had upper teeth been extracted when advised (1942) probably the lower teeth (in occlusion with upper denture) could have been retained for a longer period. The importance of the conservation of the remaining alveolar bone for denture retention was ignored by the patient. He had a "complex" about dentures, and always procrastinated, but was finally forced to have the extractions. Unfavorable prognosis substantiated.

CASE 4



INITIAL



LAST

History: 1930 Female, age 49. Gingival bleeding for several years on brushing teeth . . . extremely nervous with general fatigue and loss of energy . . . medical report showed slight anemia.

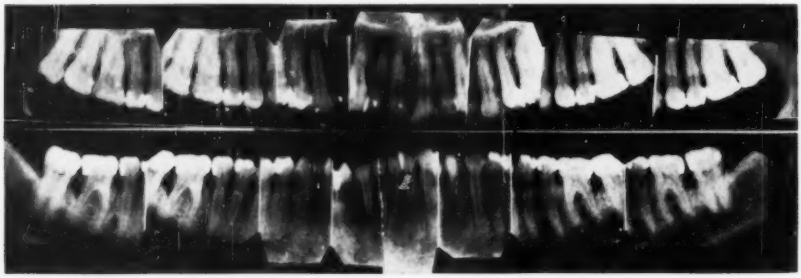
Examination: 1930 Gingivae inflamed and bluish, cyanotic color . . . supra-gingival calculus on lower anteriors and upper molars and generalized subgingival deposits . . . many deep vertical pockets with suppuration around $\frac{|\,2\ 3}{6\ 4\ |\ 2\ 6}$. . . bifurcation involvement $\frac{7\ 6\ |\ 6}{|\ 6}$. . . excessive stress and mobility $\frac{5\ 4\ |\ 1}{|}$. . . apical rarefaction $\frac{5\ |}{|}$.

Treatment: 1930 Extraction of upper right second bicuspid and lower third molars . . . subgingival curettage . . . gingival resection on lower left first molar . . . instruction in mouth care . . . periodic prophylaxis.

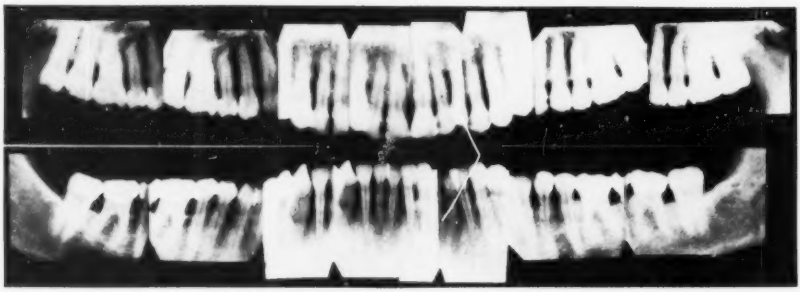
Results: 1949 No further bone resorption and restoration of cortical plate in many areas . . . gingival condition improved, tissues pink—no bleeding or suppuration except lower left first molar where recurrent inflammation and suppuration necessitated extraction (1940) . . . upper right first bicuspid extracted due to pulp degeneration. The symptoms of fatigue were greatly reduced as a result of periodontal treatment, but pernicious anemia developed (1931) and patient remains under medical care for this condition.

Comment: The maintenance of these teeth in good functional condition over this period of 11 years without any recurrence of inflammation (except as noted) can, in a large degree, be attributed to the patient's excellent home care and coöperation.

CASE 5



INITIAL



LAST

History: 1940 Female, age 32. Gingival bleeding for several years when brushing teeth . . . five years of orthodontic treatment . . . frequent anemia . . . cholecystectomy . . . two miscarriages . . . highly nervous . . . hypersensitive tissue reaction . . . optimum dietary intake.

Examination: 1940 Gingivae inflamed, mostly dark red color . . . many edematous areas . . . septal hypertrophy $\frac{3\ 2\ |}{|\ 1\ 2\ 3}$ . . . many deep vertical pockets with suppuration and bluish glossy appearance

of gingivae . . . pocket depth from 5 to 9 mm. . . . supragingival calculus on lower incisors and a generalized subgingival calculus . . . bone resorption more extensive in lower arch . . . several open contacts with food impaction . . . excessive stress with mobility of posterior teeth in lateral excursions . . . mal-occlusion with irregularity of lower anteriors and an open bite . . . good root length . . . deciduous root fragments between both lower second bicuspids and first molars.

Treatment: 1940 Subgingival curettage under local anesthesia . . . gingival resection of hypertrophic tissue . . . excessive stress relieved by selective spot grinding . . . open contacts $\frac{|\ 6\ 7}{|\ 6\ 7}$ closed by restorations . . . deciduous root on right side removed . . . instruction in mouth care . . . periodic prophylaxis.

1945 Patient agreed to extraction of upper right third molar.

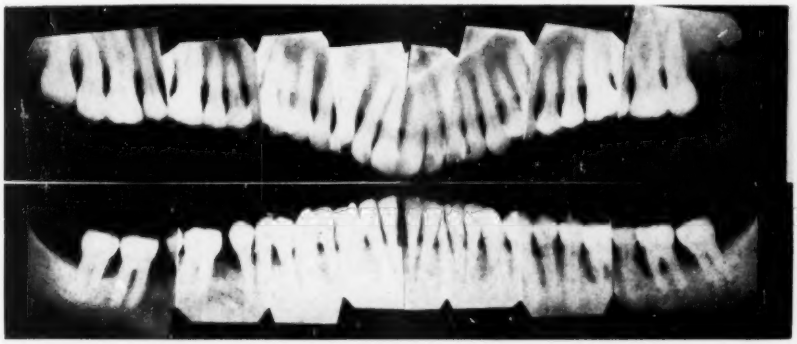
Results: 1945 Acute pericemental abscess, adema, suppuration lingual $\frac{\quad}{3\ |}$ (tendency for food impaction because of rotated position).

1946 No additional loss of alveolar bone . . . repair of bone in areas $\frac{\quad}{6\ |\ 2\ 3\ 6\ 7}$ . . . reduction in pocket depth . . . no gingival inflammation . . . roentgenograms show some bone resorption in lower right cuspid area as a result of acute inflammation (1945) . . . closure of open contact $\frac{\quad}{5\ 6\ |}$ occurred following spot grinding . . . improvement in physical condition.

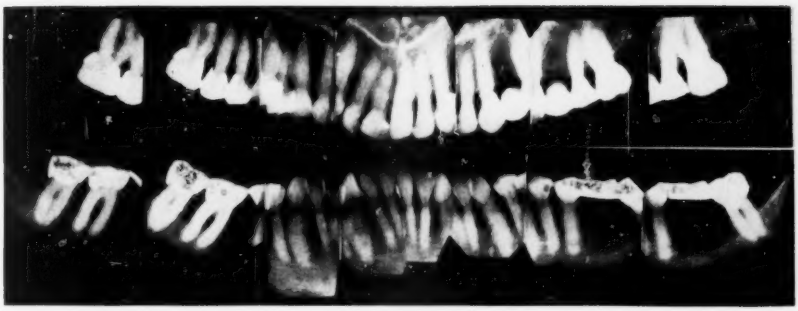
1949 Recent examination showed gingival tissues in excellent condition with no recurrence of inflammation around lower cuspid . . . pocket depth satisfactory.

Comment: Local etiologic factors included calculus, poor mouth hygiene, food impactions, open contacts, malocclusion, excessive stress and lack of function in anterior region. Predisposing systemic factors were anemia and a poor physical condition.

CASE 6



INITIAL



LAST

History: 1921 Female, aged 55. Gingival tissue bleeding upon brushing . . . arthritis (physician and dentist advised extraction of all teeth.)

Examination: 1921 Gingivae inflamed, edematous, hemorrhagic, bluish color . . . heavy supragingival calculus on lower incisors, subgingival calculus throughout . . . mobility $\frac{7 \mid \quad \mid 7}{1 \mid 1\ 6}$. . . . generalized bone resorption and pocket formation . . . excellent root length and alveolar bone structure . . . (elongation of upper teeth in original pictures).

Treatment: 1921 Extraction of $\frac{7 \mid 7}{\mid 6}$. . . . subgingival curettage . . . mouth care instruction . . . periodic prophylaxis.

1935 Extraction of $\frac{8 \mid 4}{\quad \mid \quad}$.

Results: 1949 No additional loss of bone . . . restoration of cortical plate in many areas . . . repair of bone $\frac{\quad \mid \quad}{7\ 5 \mid 3\ 7}$ (latest pictures over-exposed) . . . caries and many gingival erosions developed in past few years . . . no recurrence of gingival inflammation or pocket formation, although heavy supragingival calculus found at each prophylaxis recall.

Comment: Arthritis still present but has not progressed to any degree . . . fixed bridge restoring $\frac{\quad \mid 6}{\quad \mid \quad}$ is the original one placed in 1921 by her dentist.

The Bottom of the Pathologic Crevice

By A. B. RIFFLE, D.D.S., *Rochester, N. Y.*

REFERENCE is frequently made in dental literature to deep, narrow periodontal pockets whose base is variable in position in relation to the pocket opening. They are described as having "the shape of an inverted funnel with an occasional cryptic enlargement at its upper narrow point," or "the shape of a cone."[1, 2, 3, 4] Allusion is even occasionally made to winding tortuous pockets, with ragged, irregular edges. Usually they are described as occurring on one side of the root.

Several years of clinical periodontal practice has failed to reveal any such a tendency in pocket formation. At times the operator thinks he is in such an area, but continued careful exploration of the crevice discloses his original error.

In confirmation, the following study of the clinging attached fibers of the periodontal membrane on 478 extracted teeth fails to disclose a single deep, narrow pathologic crevice confined to a single surface, or one whose base was variable in position to the pocket opening.

Other observations, such as the characteristic patterns formed by these clinging periodontal fibers and the relative frequency with which these patterns occurred, the progressiveness of the detachment with increasing age, the average depth of the crevice in different age groups, the frequency with which the bottom of the crevice was deeper on one surface than on the other surfaces, the areas on which the deepest crevice is usually found, and the variations in this characteristic, form an interesting and important picture to the periodontist.

THE METHOD OF STUDY

Four hundred and seventy-eight teeth were used—routine extractions from the offices of 3 oral surgeons. Each tooth was recorded

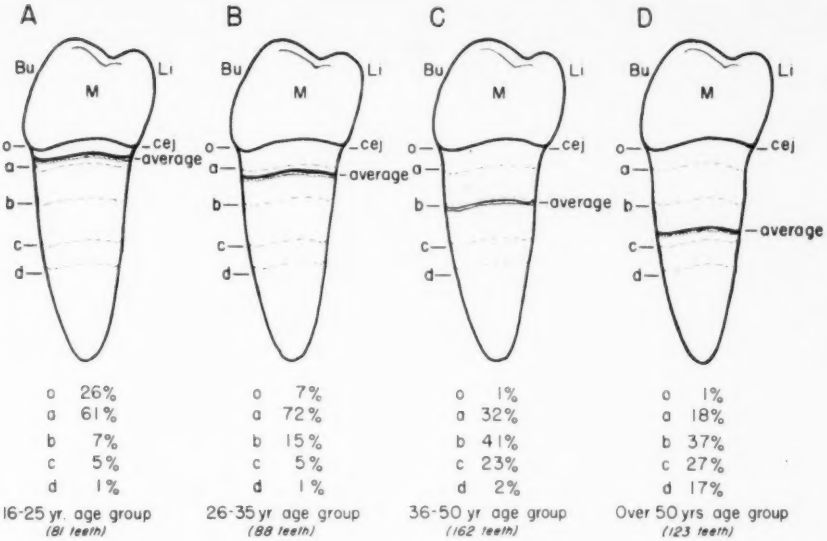


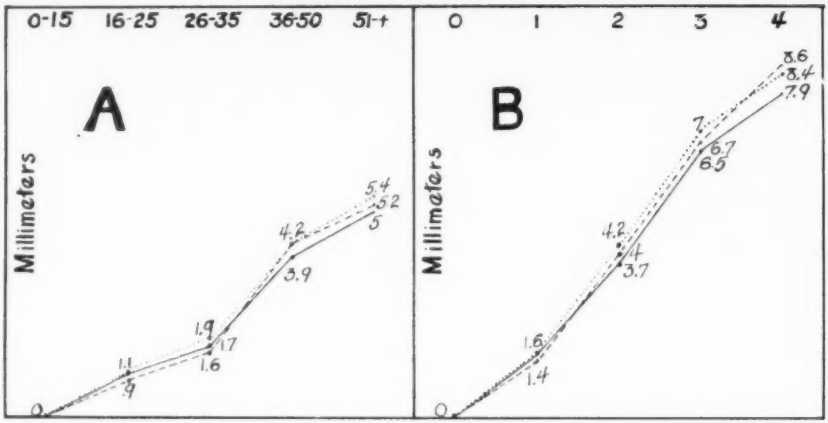
Fig. 1. Incidence and progressiveness of loss of periodontal fiber attachment with increasing age.

as to the age and sex of the patient and the reason for extraction.

Though the true bottom of the crevice in the mouth is at the line of the epithelial attachment, the clinging shreds of the periodontal membrane were considered the bottom of the crevice. As it is our practice at the chair to attempt the removal of the entire epithelial attachment during curettage, and as sometimes no epithelial attachment is found at the bottom of the crevice,[5] we believe these attached fibers are the true bottom of the crevice. This will not invalidate our observations, however, for Bass and Fullmer[6] have recently demonstrated that, after leaving the enamel, the epithelial attachment is usually a narrow band approximately paralleling these fibers.

Each tooth was measured from the cemento-enamel junction to the line of the clinging attached fibers of the periodontal membrane, and from the cemento-enamel junction to the apex, on each of its four surfaces. Recordings were made to the nearest 0.5 mm. The teeth were then arranged according to age and the observations charted and graphed. They were also rearranged according to the depth of the pathologic crevice in an arbitrary classification as described in the legend accompanying Graph 1. The deepest crevice classification was considered the classification for the entire tooth. For example, slight detachment of the fibers from the cemento-enamel junction on only one surface was sufficient to class the entire tooth in the 1 Crevice Class.

The incidence of and the progressiveness of the loss of the periodontal fiber attachment with increasing age is illustrated in Figure 1—A, B, C, and D. Before 15 years of age, only 1 tooth in 24 was found with the fibers apical to the cemento-enamel junction, and this so slight on the buccal surface as to be doubtful (not illustrated). Not so, however, in the 16-25 year grouping (Figure 1-A), for even at this early age we found only 21 of the 81 teeth (26%) free from some loss of the periodontal membrane attachment at the cemento-enamel junction (Figure 1-A O)—not necessarily on all surfaces of every tooth, but on one surface or



Graph 1 The average distance in millimeters of the periodontal fiber attachment (pf) from the cemento-enamel junction (cej) when extracted teeth are arranged according to age (A) and according to an arbitrary classification (B) as follows: 0—pf at cej; 1—pf apical to cej (1 or more surfaces) but less than ¼ the distance from cej to apex; 2—pf ¼ or more, but less than ½ cej apex; 3—pf ½ or more but less than ¾ cej to apex; 4—pf over ¾ distance cej to apex.

more, with the buccal usually the deepest. Surely a loss of periodontal fiber attachment at this early age indicates pathology, and, if so, this means that 73% (Figure 1-A 1, 2, 3, 4) of these 81 teeth had already suffered from sufficient periodontal disorders that the root surface was invaded before the age of 25. By 35 (Figure 1-B 1, 2, 3, 4) the incidence has risen to 94%; by 50 (Figure 1-C 1, 2, 3, 4) to 98%; and over 50 (Figure 1-D 1, 2, 3, 4) to 99%.

Continuous passive eruption? There was only one tooth in the 123 teeth extracted from patients over 50 years of age (Figure 1-D O) with the periodontal fibers still at the cemento-enamel junction, and 2 teeth from the 36-50 group (Figure 1-C O), but these 3 teeth throw doubt on the passive eruption theory.

One patient has been observed in whose mouth the bottom of the crevice is still on the enamel on most teeth at 50 years of age. She is not free from gingival pathology but highly resistant to its ravages. Few other patients past middle life can boast of many teeth on which this is true. We interpret the detachment of the periodontal fibers from the cemento-enamel junction as pathologic or the result of previous pathologic changes, at all ages, and consider the above findings on extracted teeth as representative of the usual clinical findings, i.e., over 94% of teeth in the mouths of patients over 35 years of age have been or are actively afflicted with periodontal pathology, and 73% of teeth are so afflicted by the age of 25.

The average depth of the crevice on the different tooth surfaces is drawn to scale in Figure 1 A, B, C, and D—*Average*, and the average measurements are shown in Graph 1-A and B. The interrupted lines in Figure 1 represent the average depth of the crevice on all teeth classified in that particular "crevice depth group" (Graph 1-B). The percentages to the left represent the frequency with which that particular "crevice depth grouping" is found in the Age Group for that specific line drawing.

Before 15 years, there is practically no fiber detachment (not illustrated). Between 16 and 25, one finds the clinging fibers on an average of 1.6 mm. apical to the cemento-enamel junction on the buccal and proximal surfaces, with the lingual surface detach-

ment lagging 0.2 mm. behind. (Figure 1-A —Average) (Graph 1-A) i.e., The loss of attachment parallels approximately the line of the cemento-enamel junction—the line from which the measurements were made—and consequently the bottom of the crevice is not a circumferential horizontal line around the root, but a line arching toward the incisal/occlusal on the proximal surfaces to correspond to the arching of the cemento-enamel junction—an average of 1 mm. on the mesial and 0.6 mm. on the distal in this series of teeth. Thus, clinically the bottom of the crevice is usually deeper on the buccal and/or the lingual, than on the proximal, in direct ratio to the height of the arch of the cemento-enamel junction on that particular tooth. This we find to be true in the mouth during curettage.

Between 26 and 35 (Figure 1-B—Average) (Graph 1 A) we find the average measurements on the different surfaces have increased slightly, but the increase is not marked, being only 0.8 mm. Again making allowance for the 1 mm. arching of the cemento-enamel junction on the mesial surface, we still find the clinging fibers, not horizontal, but arching, with the deepest clinical crevice on the buccal and/or the lingual. This also we find true in the mouth.

The greatest change takes place during the period from 36-50 years. (Figure 1-C—Average) (Graph 1 A) During this 15 year interval the average loss of soft tissue attachment is twice that of the 10 year intervals preceding it. It is as if the process had accelerated during these years. This also seems to correspond with clinical experience —an increasingly rapid loss of tissue and in pocket depth after 35.

After 50 years of age (Figure 1-D—Average) (Graph 1 A) the tissue detachment takes place less rapidly, though the percentage of teeth in the deeper crevice groupings increases. Note, however, that even in this group, 56% of these teeth had tissue loss less than ½ the distance from the cemento-enamel junction to the apex, despite the fact that 66% were extracted solely as the result of periodontal involvement, and another 20% because of periodontoclasia and dental caries.

Pocket depth is not the only criterion necessitating extraction, for many teeth remain firm and clinically comfortable with more than ½ the supporting structures gone.

From the above, and glancing again at the line drawings in figure 1 one can picture the general progress of periodontal fiber loss —the advance of the periodontal pocket—through life. Thus, in the 10 years between 16 and 25 there is an average loss of 1 mm. on all surfaces; during the next 10 years there is a loss of another 0.8 mm.; during the next 15 years, 2 mm.; after 50, another 1 mm. The average for all ages show a deeper pathologic crevice on the buccal and/or the lingual than there is on the mesial and/or the distal, deeper on the buccal in early life, just as deep or deeper on the lingual in later life. The proximal was rarely deeper than the deepest part of at least one of the adjoining surfaces. This will be discussed under the variations in crevice depth.

Contrary as these findings are to the usual teachings, the author has found them to be consistently true at the chair.

VARIATIONS IN CREVICE DEPTH ON DIFFERENT SURFACES OF THE SAME TOOTH

The bottom of the crevice is not necessarily at the same distance from the cemento-enamel junction on all surfaces of the same tooth, as the average recordings above might lead us to believe. Neither is a large variation common.

Chart 1 shows the average per cent of variation. Thus, 75% of the 478 extracted teeth had variations in depth of only 2 mm. or less between the shallowest to the deepest lines of clinging periodontal fiber attachment (1st 3 columns—all teeth) and 88% have variations of only 3 mm. or less (1st 4 columns—all teeth). Half of the remaining teeth show variations of only 4 mm., and only 6% 5 mm. or over. There

CHART I.

Age Groups	Per Cent of Variations in Crevice Depth						
Years	0 mm	1 mm	2 mm	3 mm	4 mm	5 mm	Over 5 mm
0-15 (24 teeth)	96	4					
16-25 (81 teeth)	33	37	16	7	2	4	
26-35 (88 teeth)	28	40	16	11	4	1	
36-50 (162 teeth)	17	30	26	13	6	4	5
Over 50 (123 teeth)	21	16	21	20	13	3	4
All Teeth (478)	27	28	20	13	6	3	3

are no over 5 mm. variations until after 35, and the tendency for these larger variations seems to increase with age.

In interpreting these findings, however, one must again take into consideration the incisal/occlusal arching of the cemento-enamel junction on the proximal surfaces (see above). The measurements were made from the cemento-enamel junction to the clinging periodontal fibers. Therefore, many teeth showing variations of 1 or 2 mm. on the chart actually present the line of attachment as a circumferential horizontal line (Figure 2-f).

Of the 478 teeth used in the entire study, 8 were discarded for obvious inaccuracies and 50 had the periodontal fibers at the cemento-enamel junction, leaving 420 teeth for the basis of the following observations.

The most common pattern (47%) formed by these clinging fibers is one in which the buccal, or the lingual, or both are apical to those found on the other surfaces (Figure 2-b, c, and d). Before 35 the buccal is usually the site of the deepest crevice; after 35, the lingual surface is just as frequent; at times both buccal and lingual are of equal depth, with the proximal surfaces definitely shallower. The actual percentages are: buccal deepest—92 teeth, or 22%; lingual deepest —78 teeth or 19%; buccal and lingual equal, but deeper than the proximal—24 teeth or 6%.

The first variation (30%) from the most common pattern is to find the periodontal fibers parallel to the cemento-enamel junction all around the root. Whether this line of fibers is a horizontal circumferential line, or whether it is a line arching toward the incisal/occlusal on the proximal depends on the height of the arch of the cemento-enamel junction as described above, i.e., arching of the cemento-enamel junction results in a pattern with arching of the fibers—no arch results in a horizontal circumferential line around the root (Figure 2-d and e).

The second variation (10%) is one in which the measurements from the cemento-enamel junction to the periodontal fibers are greater on the proximal than on the buccal and/or lingual. Characteristic of this group is a high arching of the cemento-enamel junction on the proximal as described above, and a resulting periodontal fiber pattern which is identical with that described in the first variation above—a horizontal circumferential line around the root (Figure 2-f).

The third variation (13%) consists of those in which the bottom of the crevice is deeper on one proximal surface than on the other. Actually there is a much larger per cent than 13 which might fall into this group, for many teeth in which the buccal and/or the lingual was considered the deepest crevice (above—the most common pattern) also presented a corresponding depth on the adjoining proximal surface or surfaces. The actual center of the most apical portion of the periodontal fiber loss was used

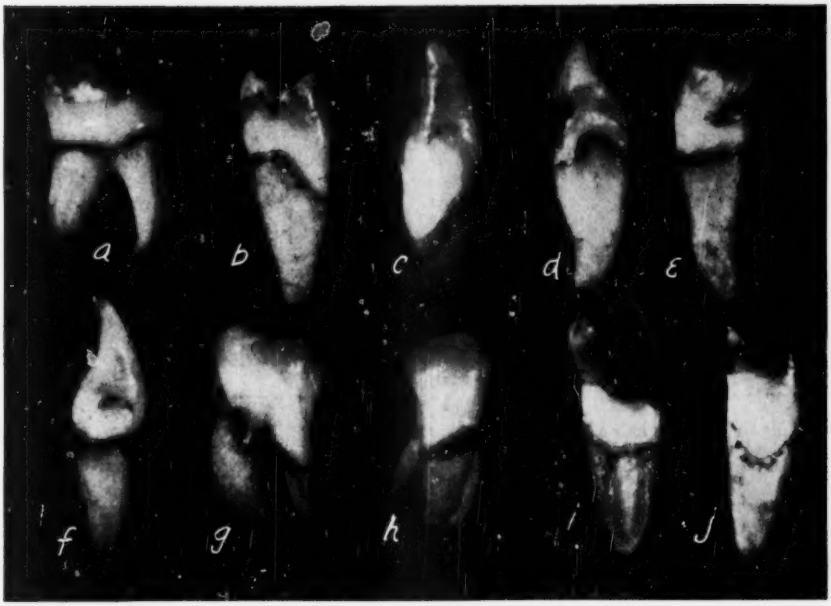


Figure 2. The 4 characteristic patterns formed by the clinging attached periodontal fibers on extracted teeth. a, fibers at cemento-enamel junction. b, crevice deepest on the buccal. c, crevice deepest on the lingual. d, crevice deeper on both buccal and lingual than on proximal. (b, c, and d together are The Most Common Pattern) e, fibers parallel to the cemento-enamel junction. (The first variation) f, fiber loss greatest on the proximal, but high arching of cemento-enamel junction results in a pattern similar to e. (The second variation) g and h, fiber loss greater on one proximal surface than on the other. (The third variation) i and j, the slight declivity toward the apex occasionally found.

as a criterion but frequently a definite distinction was difficult. Separate recordings of the mesial and distal surfaces showed no significant difference between the two in the frequency with which the deepest crevice occurred (Figure 2-g and h).

On only 3% of all the teeth were found a slight "dipping" of the fibers toward the apex on the proximal (Figure 2-i and j). Rarely did this apical declivity exceed 1 mm. and could not possibly be confused with the deep "vertical" pockets of the literature.

Statistical study of a large number of x-ray negatives would undoubtedly show a larger per cent of crevices with deeper bone loss on one proximal surface than the other, than is shown in the Third Variation. This would be explained in one of two ways: First, the buccal and/or the lingual would be as deep or deeper, but the bottom of the crevice would be obscured by the shadow of the root; Second, bone loss could exceed actual soft tissue detachment, giving a false picture of the crevice depth on the x-ray negative.

There is a definite practical application to the above findings during subgingival curettage. Note that 87% fall into a clinical category in which the tissue loss on the buccal and/or the lingual is deeper than or equal to the proximal. (Most common pattern, 1st variation, and 2nd variation) 13% have the deepest measurement of tissue loss on the proximal surface (3rd variation), but on these, one or both of the adjoining surfaces (buccal and/or lingual) will present the bottom of the crevice at an equal depth. Only 3% present a slight "dip" toward the apex on the proximal surfaces. Deep losses of periodontal fiber attachment were never found on the proximal surfaces alone.

Therefore, access to the deepest portion of the bottom of the crevice during curettage should be possible in at least 97% of teeth from the buccal and/or the lingual. On the remaining 3%, the slight apical concavity formed by the fiber loss should present little difficulty during instrumentation.

Large variations between the deepest point of detachment and the shallowest were few. Only 19 of the 478 teeth showed statistical variations of 5 mm. or more (Chart 1). Of these, 5 were obvious inaccuracies and 6 accounted for the seeming large variation by unusually high archings of the cemento-enamel junction on the proximal surface, presenting the patterns described above.

This left only 8 teeth of the 478 on which a deep narrow pocket was possible. On 5 of these, though there were rather deep variations in the depth of the crevice on opposite sides of the tooth, the pattern was similar to those in Figure 2-b, c, g and h—not a narrow, funnel shaped one. Rather, the pattern presents a diagonal tapering over the adjoining surfaces from the shallowest to the deepest part of the crevice.

Of the 3 remaining teeth, 2 simulate a possible deep narrow crevice (Figure 2-j); the 3rd was probably a forcep injury to the remaining attached fibers.

With the above in mind all 478 teeth were reviewed and just a few found in which the change in the line of the pattern from one surface to another was more or less abrupt—approaching a right angle—instead of the usual straight or gradual curving so characteristic. There were not enough, however, nor were they characteristic enough to justify a special classification and description.

None of these answer the descriptions of the deep narrow pockets of the literature, and the following conclusion seems justified: When the remaining attached fibers of the periodontal membrane are used as the bottom of the crevice on extracted teeth, the deep, narrow funnel shaped pocket on one side of the ropt either does not exist, or exists in such small numbers as to be negligible.

CONCLUSIONS

A study of the pattern formed by the clinging fibers of the periodontal membrane on 478 extracted teeth showed:

1. A progressively increasing depth of the pathologic crevice with increasing age, most marked between 35 and 50 years of age.
2. An incidence of loss of the periodontal fiber attachment beyond the cemento-enamel junction in 73% of teeth before 25 years of age and 94% by 35 years.
3. Continuous passive eruption is questioned because a few teeth in the older age groups still present the clinging attached periodontal fibers at the cemento-enamel junction.
4. Pocket depth is not the only criterion for extraction.
5. The 4 characteristic patterns formed by the loss of the soft tissue attachment are described and discussed.
6. The pathologic crevice is usually deeper on the buccal and/or lingual than on the proximal surfaces.
7. Deep detachment of the periodontal fibers was never found on the proximal surfaces alone.
8. No deep narrow funnel shaped crevices whose bases were variable in relation to the pocket opening were found.

BIBLIOGRAPHY

1. Black, G. V., *The American System of Dentistry*. Volume 1. Lea Brothers and Co., 1886, pp. 969-971.
2. Prinz, Hermann and Greenbaum, Sigmund S. *Diseases of the Mouth and Their Treatment*. Second Edition. Lea and Febiger, 1939, pp. 127 and 139.
3. Box, Harold Keith. *Treatment of the Periodontal Pocket*. University of Toronto Press, 1928, p. 9.
4. Thoma, Kurt H. *Oral Pathology*. C. V. Mosby Company, 1941, pp. 718-719.
5. Goldman, Henry M. *Periodontia*. C. V. Mosby Company, 1942, p. 281.
6. Bass, Charles C. and Fullmer, Harold M. The Location of the Zone of Disintegrating Epithelial Attachment. *Jour. Dent. Research*, 27:623: Oct., 1948.

The Gingival Structures in Diabetes Mellitus*

By HAROLD G. RAY, D.D.S., M.S.** AND BALINT ORBAN, M.D., D.D.S.***

THE INFLUENCE of systemic diseases on the periodontal tissues has long been a major problem in stomatology.

There has been a persistent clinical impression that periodontal diseases are increased in severity in patients with diabetes mellitus and that in this condition the resistance of the gingival tissues is lowered in its reaction to local irritations, thereby increasing the inflammatory process[1,2,4,7,8,-11,12,13,14,23]

The purpose of the present investigation was to study by histological examination the gingival tissues taken by biopsy from patients with known diabetes mellitus and to compare them with patients who were non-diabetic.

REVIEW OF LITERATURE

There are a number of reports in the literature based on clinical observation of the gingiva of diabetic patients. However, only two reports have been published on histologic findings.

Histologic study of human material. Ziskin, et al.[25,26] made a histological study of the gingiva in nineteen juvenile and adult diabetics who were controlled either by insulin or by diet. They reported hyperplasia of the epithelium and connective tissue, hyperkeratinization of the epithelium, and perinuclear vacuolization of the prickle cells. There was an increase in the number of fibroblasts and in the thickness of the collagenous fibers. The capillaries were increased in number and showed swelling of the endothelial cells.

In most cases the inflammatory exudate was either absent or slight. In cases where it was marked, the response appeared to be less than would be expected on the basis of the gross pathologic changes of the gingiva. By special staining they found that the deposits of glycogen granules were greater in the gingival specimens of the diabetic patients than in those of nondiabetic persons. The granules were scattered throughout the epithelium and were seen in small numbers in the reticular layer of the lamina propria. These authors pointed out the possibility that some of the gingival changes that they observed were a result of the nutritional and metabolic imbalances of the diabetic state.

Experimental study of animal material. Glickman[6] conducted an experimental study on 103 albino rats which were injected with alloxan to produce alloxan diabetes. A group of eighteen animals were maintained as controls. He studied the histologic changes in the pancreas and kidney and particularly those in the periodontium. No gross pathologic changes were found in the oral cavities of either the control or diabetic animals. Certain histologic findings in the oral tissues of the control group as well as those animals with chronic diabetes were described. Glickman concluded that the changes observed in the diabetic animals appeared with comparable frequency and severity in the control group, and that the changes were caused by local influences.

MATERIAL AND METHODS

Material: The present study is based on the histologic investigation of gingival biopsy specimens of seventy-six patients.

Thirty of the patients were adults under treatment for diabetes mellitus in the Metabolic Clinic of the University of Illinois Research and Educational Hospital and were

From the Department of Histology, College of Dentistry, University of Illinois and part of a dissertation submitted in partial fulfillment of the requirements for the degree of Master of Science in Histology in the University of Illinois. Aided by a grant from the Division of Research Grants and Fellowships of the National Institute of Health, United States Public Health Service.

**From the Division of Dental Medicine, College of Dentistry and the George Williams Hooper Foundation for Medical Research, University of California, San Francisco, California.*

***From the Colorado Dental Foundation, Colorado Springs, Colorado.*

selected at random. These patients ranged in age from twenty-five to seventy-one years and the duration of diabetes was from three months to ten years. The control of the diabetes of these patients was determined according to the severity of glycosuria. Six were in good control, eleven in fair control, and thirteen in poor control.

Six of the patients were juveniles under treatment for diabetes mellitus in the Pediatric Clinic of the University of Illinois Research and Educational hospital. These patients ranged in age from four years to twelve years.

Gingival biopsies were taken from thirty adults who were nondiabetic and were inmates of the Chicago State Hospital at Dunning, Illinois. These patients ranged in age from twenty-four years to seventy-two years and were physically healthy individuals with the exception of five who were suffering from particular illnesses.

Gingival biopsies were also obtained from ten juvenile non-diabetic patients. Six of these were patients of the Pediatric Clinic who were not under treatment for diabetes mellitus, and four were in good health. They ranged in age from four years to twelve years.

All of the patients were classified on the basis of clinical examination according to the degree of gingivitis. The diabetic patients were also classified according to the control of the disease.

Methods: Each biopsy specimen was an interdental papilla and included part of the attached gingiva. The tissue was excised under local anesthesia which consisted of one-half to two cubic centimeters of a two per cent novocain solution without epinephrine. This solution was injected into the mucobuccal fold to avoid alteration of the tissue to be examined.

The specimens were immediately fixed in Zenker's formalin, some in ninety-five per cent alcohol, and imbedded in paraffin. The specimens were sectioned labiolingually so as to include the pocket epithelium (Fig. 1). The specimens were stained with hematoxylin-eosin, Mallory's connective tissue stain, Gomori's, Bauer-Feulgen's, and Best's carmine stain for glycogen. Special stains, such as Mayer's mucicarmine and Weigert's elastic tissue stain were used on selected sections of the specimens from adult and juvenile diabetic patients, as well as a comparable number of non-diabetic adults and children.

Fig. 1. Photomicrograph showing the gingiva of a case of diabetes (Case No. 47, age 50, 10 years duration, fair control). Note the smooth surface, lack of hornification and accumulation of inflammatory cells at the ends of the epithelial ridges of the attached gingiva (I).
Hematoxylin-eosin stain.

The histological observations were tabulated for each specimen (Table 1).

FINDINGS

I. *Clinical Observations.*

The specimens of adult diabetic patients were classified into groups of mild, moderate and severe gingivitis. None of the diabetic patients were free from gingivitis. The control specimens taken from adult nondiabetic

TABLE 1
Summary of Histologic Findings in the Gingiva of Diabetic and Nondiabetic Patients

Histologic Findings	*Diabetic*				*Nondiabetic*			
	Severity of Gingivitis							
	Mild	*Mod.*	*Severe*	*Total*	*Mild*	*Mod.*	*Severe*	*Total*
Number of Biopsies Examined	19	14	3	36	23	14	3	40
I. Epithelium								
a. Surface Character								
Stippled	8	4	0	12	12	9	0	29
Smooth	11	10	3	24	3	5	3	11
b. Keratinization								
Hornified	6	1	1	8	14	7	0	21
Parakeratotic	13	11	1	25	7	4	1	12
Unhornified	0	2	1	3	2	3	2	7
c. Vacuolization of Cytoplasm								
1*	14	4	1	19	16	8	2	26
2**	1	9	2	12	1	1	1	3
3***	3	0	0	3	0	0	0	0
d. Intranuclear Vacuolization	7	5	1	13	0	2	1	3
e. Invasion of Attached Gingiva by Lymphocytes	9	5	0	14	4	6	3	13
II. Lamina Propria								
a. Inflammation								
0	5	0	0	5	8	5	0	13
1*	12	3	0	15	10	7	0	17
2**	0	7	1	8	3	1	2	6
3***	2	4	2	8	1	1	2	4
b. Areas of Connective Tissue Change	11	5	1	17	6	3	0	9
c. Areas of Necrotizing Plasma Cells	16	13	3	32	17	7	3	27
d. Foreign Bodies	6	4	0	10	4	3	0	7

0—No appreciable occurrence
*—Mild occurrence
**—Moderate occurrence
***—Severe occurrence

patients were selected in comparable numbers for each group. All the juvenile diabetic and nondiabetic patients presented a mild gingivitis.

Mild gingivitis was characterized by a slight reddening of the marginal gingiva occurring only in certain areas of the gingiva. Moderate gingivitis was defined as a condition in which generalized reddening of the marginal gingiva occurred with slight blunting of the interdental papillae. Severe gingivitis was designated as the condition in which diffuse reddening and swelling of the marginal gingiva and severe blunting of the papillae occurred with accompanying hemorrhagic tendencies.

No correlation between the control of the diabetic state and the clinical severity of gingivitis was observed. In the thirteen cases of mild gingivitis in diabetics, six were in

poor control, five in fair, and two in good control. In the moderate gingivitis group four were in poor control, six in fair, and four in good control. However, in the group having severe gingivitis all were in poor control.

II. *Histologic Findings:*

1. *Epithelium.*

a. *Surface Character.* The number of specimens from adults showing a lack of stippling and thus a smooth surface of the attached gingiva increased with the severity of gingivitis (Fig. 1). This increase occurred in both the diabetic and non-diabetic patients (Table 1) but was greater in the diabetic group. Sixty-four per cent of the diabetic patients and twenty-seven per cent of the non-diabetics showed a smooth surface of the attached gingiva.

Each of the six cases of juvenile diabetes, all of whom showed clinically a mild gingivitis, presented a smooth surface of the attached gingiva. In the ten control non-diabetic juveniles two showed a smooth surface.

b. *Keratinization of Epithelium.* Hornification of the attached and free gingiva was found more consistently in the non-diabetic than in the diabetic patients (Table 1).

Instances of hornification tended to decrease, while those of parakeratosis tended to increase with the severity of gingivitis. The epithelium was hornified in fifty per cent of the adult nondiabetic patients and in only twenty per cent of the adult diabetic patients. A similar difference in the incidence of hornification was observed in the juvenile diabetic and non-diabetic group.

c. *Vacuolization of the Cytoplasm.* Vacuolization of the cytoplasm of the epithelial cells was found to occur almost regularly in the specimens of both the diabetic and non-diabetic patients. This change occurred in varying degrees. The degree of occurrence was, therefore, evaluated by 1 plus for scattered occurrence, 2 plus for medium occurrence, and 3 plus for a generalized occurrence throughout the epithelium.

The adult diabetic and non-diabetic patients of the mild and severe gingivitis group showed the same incidence and degree of this vacuolization. The group with moderate gingivitis, however, showed the diabetic patients to have an increase in degree of occurrence over that of the non-diabetic persons.

The specimens from juvenile diabetic patients showed vacuolization of the cytoplasm in each of the six of the specimens, two of which were 3 plus in degree and the remaining four were 1 plus.

The control juvenile non-diabetic patients showed vacuolization of the cytoplasm in only two of the specimens and these were of a degree of 1 plus.

There appeared to be a slight increase in the incidence of vacuolization of the cytoplasm in the diabetic patients as compared with the non-diabetic patients (Table 1).

d. *Intranuclear Vacuolization of Epithelial Cells in the Germinative Layer.*

In the germinative layer of the epithelium a change in the nucleus of the epithelial cells was observed. This appeared as a condensation of the nuclear material at the nuclear membrane, leaving the center of the nucleus clear (Fig. 2).

The incidence of intranuclear vacuolization was greater in the adult diabetic patients than the adult non-diabetic cases (43% and 10% respectively). In most of

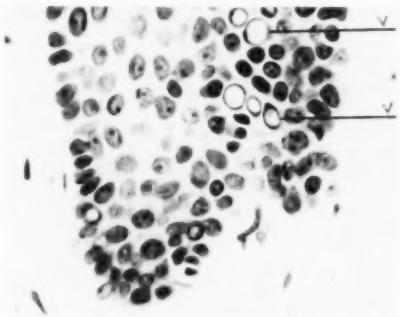


Fig. 2. Photomicrograph, high magnification, showing intranuclear vacuolization of basal cells (V). Note the condensation of chromatin material at the nuclear membrane (Diabetes Case No. 42, age 35, 6 months duration, poor control). Hematoxylin-eosin stain.

the instances in which this change occurred in the diabetic cases the entire nucleus was degenerated and what appeared to be the nucleolus was packed against the nuclear membrane giving a signet ring appearance. This finding was not seen in the non-diabetic cases. However, in three instances changes were seen which slightly resembled those observed in diabetes.

The gingival epithelium of juvenile patients, diabetic as well as non-diabetic, showed no intranuclear vacuolization.

e. *Invasion of Epithelium of attached Gingiva by Lymphocytes.* All gingival biopsy specimens showed invasion of the epithelium lining the sulcus by neutrophil leucocytes and lymphocytes. The epithelium of the attached gingiva is rarely invaded by neutrophil leucocytes, but with the increase in inflammation in the papillary layer of the lamina propria it is often invaded by lymphocytes.

The adult diabetic and non-diabetic patients showed no difference in the incidence of invasion of the epithelium of the attached gingiva by lymphocytes.

There was, however, a marked difference in the juvenile group. The epithelium of the attached gingiva of all juvenile diabetic patients showed invasion by lymphocytes, whereas invasion was observed in only one instance in the non-diabetic juveniles.

f. *Glycogen Content.* No difference was observed between the gingival biopsy specimens of diabetic and non-diabetic patients stained with Gomori's, Bauer-Geulgen's glycogen stain and Best's carmine glycogen stain. Glycogen granules were seen deposited in the cytoplasm of the cells of the outer third of the epithelium. A quantitative analysis was not made.

2. *Lamina Propria.*

a. *Inflammation.* All specimens showed inflammation of the free gingiva, especially at the site of the sulcus (Fig. 1). This inflammation was characterized by accumulation of plasma cells, lymphocytes and polymorphonuclear leucocytes.

Inflammation of the attached gingiva was frequently observed, but not with the same prevalence as in the free gingiva. The inflammatory cells in the attached gingiva were predominantly plasma cells and lymphocytes but polymorphonuclear leucocytes were occasionally seen.

The size of the inflammatory area in the attached gingiva varied. In order to evaluate the difference in severity of inflammation the extent of the inflammatory areas and the concentration of inflammatory cells were recorded by 0 for no appreciable, 1 plus for scattered, 2 plus for medium and 3 plus for generalized occurrence throughout the lamina propria (Table 1).

In both diabetic and non-diabetic patients the severity of inflammation of the attached gingiva increased with the degree of gingivitis observed clinically. In the groups showing clinically moderate gingivitis the inflammation seemed to be more severe in the adult diabetic patients than in the non-diabetics.

In moderate and severe gingivitis, inflammatory cells were frequently seen in the papillary layer of the attached gingiva of the diabetic patients, but only rarely in the non-diabetic patient (Fig. 1).

There was no correlation between the severity of inflammation and the control of diabetes.

b. *Changes in the Structure of Connective Tissue.* A number of specimens presented a peculiar change in the structure of the connective tissue in certain areas (Fig. 3). These occurred most frequently in the deeper portions of the reticular layer of the lamina propria in areas free of inflammation. These changes were characterized by pyknosis of the nuclei of the fibroblasts and a breaking up of the collagenous fibers. The ground substance, in particular, seemed to have undergone alterations which made the whole area appear as an eosin-staining granular mass imbedded in a basophilic material. The areas were peculiarly free of capillaries and arterioles (Figs. 3 and 4).

Mallory's connective tissue stain showed the areas of fragmented collagenous fibers as light blue granules. The possible presence

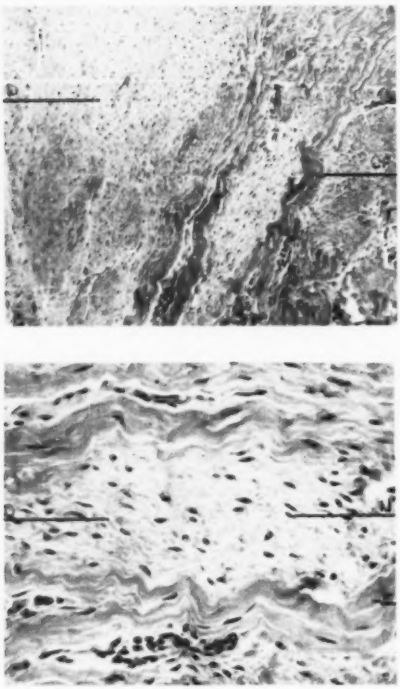


Fig. 3. Photomicrographs showing A, low magnification and B, high magnification of change in the structure of dense connective tissue (D) in lamina propria. (Diabetic Case No. 70, age 23, 1 year duration in fair control). Note pyknosis of nuclei of fibroblasts (X) and bundles of normal collagenous fibers (CF).

Hematoxylin-eosin stain.

of mucin in the areas of connective tissue breakdown was suggested by the pink to red staining with Mayer's mucicarmine stain.

The incidence of areas of connective tissue change was higher in the diabetic (43%) than in the non-diabetic patients (23%). This connective tissue alteration was found in patients with each degree of gingivitis.

Practically all of the diabetic patients who showed these changes were in only fair or poor control. Of the nine non-diabetic patients who were affected two had a history of epileptic seizures, one suffered from subacute bacterial endocarditis, one had a brain tumor, and one had a history of renal disease.

c. *Areas of Necrotic Plasma Cells.* Groups of dead and dying plasma cells were observed in areas of chronic inflammation in almost all of the specimens studied (Fig. 5). These areas were characterized by accumulations of plasma cells in all stages of disintegration showing pyknosis of their nuclei, karyorrhexis and karyolysis, and were frequently accompanied by intercellular and intracellular edema. The final result of the disintegration of these cells was the appearance of granules of varying size which stained poorly or polychromatically with hematoxylin-eosin[21]. These granules did not resemble the hyalin globules known as Russell's bodies which were also seen occasionally in these areas.

There was no difference in the frequency of this finding in the diabetic and non-diabetic patients.

d. *"Foreign" Bodies in the Connective Tissue.* Foreign bodies of different natures were found in the connective tissue of the gingiva of both diabetic and non-diabetic patients (Table 1).

The majority were of oval or spherical shape. Some had an irregular outline (Fig. 6). Most of these bodies stained light to dark pink with eosin, some stained intensively blue with hematoxylin and two stained blue only in the center. These staining reactions suggested different degrees of calcification. The "foreign bodies" were either single or arranged in groups. In one specimen they occurred in a rather long row which extended close to the gingival margin, and appeared as hyalinized rings with some necrotic material in the center.

Most of these bodies were surrounded by one or two layers of spindle-shaped connective tissue cells. In one instance foreign body giant cells were found.

In three instances striated rectangular bodies were found which stained blue with hematoxylin, and appeared to be fragments of cementum from the root surfaces (Fig. 4). One calcified body was a phlebolith.

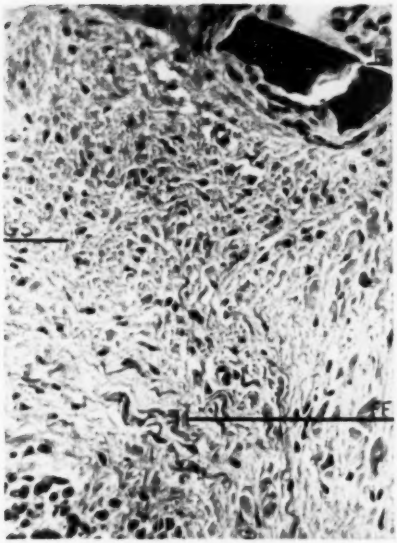


Fig. 4. Photomicrograph, high magnification, showing area of change in the structure of connective tissue in a juvenile diabetic (Case No. 3, age 13, 4 years duration, poor control). Note granular appearance of background (G.S.), fragmented collagenous fibers (FF) and presence of piece of fractured cementum (FC).
Hematoxylin-eosin stain.

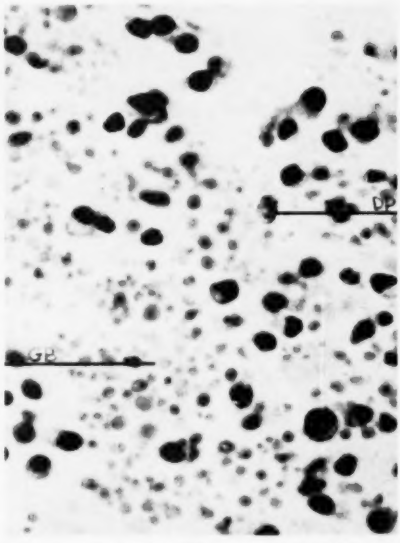


Fig. 5. Photomicrograph, high magnification, showing area of necrotic plasma cells in the lamina propria. (Diabetic Case No. 45, age 71 years, poor control). Note granular bodies (GB) which stain polychromatically and degenerating plasma cell (DP).
Hematoxylin-eosin stain.

DISCUSSION

Analysis and comparison of the histologic findings in the gingival biopsies of patients with diabetes mellitus and of nondiabetic patients showed that none of the changes observed could be designated as characteristic for the gingiva of the diabetic patient. Glickman[6] came to a similar conclusion on the basis of his findings in alloxan diabetic animals.

Most of the changes were related to the severity of gingivitis and as such probably represented tissue reaction to local irritation. Some of these reactions appeared to be accentuated in the diabetic patients. None of the diabetic patients were free of clinical gingivitis. On the other hand, a cross section of a population of similar age distribution showed thirty to forty per cent incidence of normal gingiva.[15] This would indicate that the incidence of gingivitis is increased in diabetes mellitus.

Change in Surface Character. The normal attached gingiva shows a wavy surface which accounts for the clinical appearance of stippling. Orban[20] has pointed out that the disappearance of stippling is an indication that gingivitis has extended into the attached gingiva. This interpretation could be confirmed by the finding, that the number of specimens showing a smooth surface of the attached gingiva increased with the severity of gingivitis. The greater incidence of smooth surfaces of the attached gingiva in the diabetic patients might indicate a higher reactivity of the gingival tissue to irritation.

Decrease in Hornification. The number of specimens showing hornification of the epithelium decreased as the severity of gingivitis increased. This change from hornification to parakeratosis or complete lack of hornification is very probably also related to the increase in severity of inflammation of the gingiva.

The diabetic cases again showed a greater number of instances in which this change occurred. The absence of hornification might also be a sign of a higher reactivity to irritation in the cases of the diabetics. We could not confirm the observation of hyperkeratinization or hyperplasia of the epithelium made by Ziskin, et al.[27,28]

Cytologic Changes in the Epithelium. Vacuolization of the cytoplasm of the surface epithelial cells is seen in many inflammatory conditions of the skin and is referred to as hydropic degeneration or intracellular edema.[16] This is also a common finding in gingivitis of any origin. The number of cases showing vacuolization of the cytoplasm was similar in our diabetic and non-diabetic patients. However, the degree of vacuolization appeared to be slightly accentuated in the diabetic patients. Ziskin, et al.,[27,28] suggested that perinuclear vacuolization of the prickle cells was the result of the diabetic state. In the specimens stained for glycogen the granules were found deposited in the cytoplasm. The possibility exists that in some cases the vacuolization may represent the loss of glycogen in the process of preparation.[26]

Intranuclear vacuolization of the basal cells was found predominantly in the adult diabetic patients. Degeneration of this type has been reported in the hepatic cells in cases of diabetes where the nucleus was found to contain glycogen,[26] but we were unable to make this observation with our method of glycogen staining. The possibility of artefact caused by superimposition of cytoplasmic vacuoles on the nucleus could be ruled out.

The possible significance of this finding in relation to the diabetic state must await further investigation.

Increase in Intensity of Inflammation. The present study showed an intensification of the inflammatory process in gingivitis of the diabetic patients as compared with gingivitis in non-diabetic persons. This was especially noted in the group of moderate gingivitis. The intensification of the inflammation manifested itself by an increase in the number of inflammatory cells in the reticular layer and by the occurrence of inflammation in the papillary layer where it is rarely seen in the nondiabetic persons. This increase in the inflammatory reaction of the connective tissue of all parts of the lamina propria and in the absence of a comparably severe surface irritation might be related to an altered metabolism resulting from the diabetic state. The findings of Ziskin and co-workers[27,28] that the inflammatory response to irritation in the gingiva of diabetics was less than could be expected from the clinical observation were contrary to our experience.

Richardson[22] concluded that in diabetes the tissues in general showed a diminished capacity to form antibodies and that an excessive local inflammatory reaction to any irritant was consequently produced. Menkin[17] observed that hyperglycemia tends to be increased in depancreatized dogs having a superimposed acute inflammation. The accentuated hyperglycemia was explained as a result of the increased proteolysis at the site of the inflammation. The local proteolysis produces local acidosis and intensification of the inflammation.

Change in the Structural Appearance of Connective Tissue. Changes in the structural appearance of the connective tissue were observed in specimens of both diabetic and non-diabetic cases, regardless of the age of the patient. The bundles of collagenous fibers were broken up and replaced by a mass of irregular granules which were suspended in a basophilic homogeneous material. In specimens stained with hematoxylin-eosin, the ground substance between the pink staining collagenous fiber particles were basophilic. Furthermore, the sections treated with Mallory's connective tissue stain tended to confirm the presence of this structural change while the sections stained with mucicarmine suggested the presence of mucin or mucin-like substances in the affected areas.[5] It is known that the principal constituents of the ground substance of the connective tissue are chondromucoid and chondroalbuminoid.[5] Mucin might thus be the end

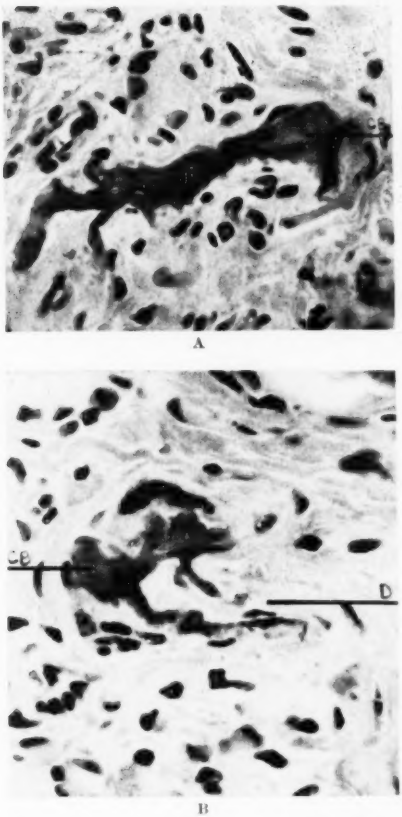


Fig. 6. Photomicrographs: A, high magnification, showing calcification appearing as calcified connective tissue (CB) (Diabetic Case No. 11, age 21 years, duration 10 years, poor control); B, high magnification, showing calcification or hyalinization of connective tissue (CB). (Diabetic Case No. 70, age 23 years, duration 1 year, fair control). Note degeneration of connective tissue (D) surrounding calcified body (CB).
Hematoxylin-eosin stain.

product observed between the broken-up collagenous fibers.

The loose connective tissue is a dynamic tissue and subject to change in response to irritants of every nature, toxic or mechanical, and as such very probably continually goes through a cycle of disintegration and repair. The change observed in the structure of the connective tissue may represent a phase of this process. A histochemical study of the nature of these connective tissue changes would no doubt help to evaluate them. Gersh[5] and others have suggested that the physical state of the ground substance of connective tissue is a state of polymerization. It may be weakly polymerized as in mesenchym of embryos, depolymerized as in oedema or highly polymerized as in cartillage or bone. The process is analogous to the way acrylic monomers may form polymers of different consistency depending on temperature pressure and so on. All substances which pass between blood vessels and cells must pass through the ground substance which contains a glyco protein.

It is not possible, at the present time, to decide whether the observed change in the connective tissue is caused by a local inflammatory reaction, mechanical irritation, a systemic factor, or a combination of these influences. Proliferation of the connective tissue elements, as reported by Ziskin[27] was not evident in this study.

Presence of Groups of Necrotic Plasma Cells. Groups of dead and degenerating plasma cells in restricted areas were found in a high percentage of both the diabetic and non-diabetic cases. Previous study of gingival biopsies from cases of gingivitis in healthy persons and patients with various diseases[21] showed a comparable incidence of such foci. These findings, therefore, are not characteristic for the gingiva in diabetes.

In some instances the degenerating plasma cell produces a hyalin-like globule in its cytoplasm which stains pink and is eventually set free to remain in the tissues as Russell's bodies. The polychromatic granules of cellular debris are quite different, however, and appear as necrotic material. Menkin[18] states that one of the substances liberated by degenerating cells in inflammation is necrosin. This substance supposedly causes cellular injury and further inflammatory reactions, which might explain the penetration of the inflammatory process into the deeper layers of the supporting tissues following the surface inflammation of the gingiva.

Foreign Bodies. Hyalin-like and calcified bodies were found in the gingiva of both

diabetic and non-diabetic patients. Calcified bodies of different origin have been described in gingival biopsies.[19] These were cementum remnants, cementicles, calcified thrombi, and crystalline structures.

The fact that "foreign" bodies were found in more than one-fifth of our cases calls for further study of their prevalence and significance in a larger number of biopsies.

The bodies we found appeared to be either remnants of cementum, cementicles, or hyalinized and finally calcified connective tissue. The remnants of cementum were derived either from incompletely shed deciduous teeth or from fractures of root surfaces.[12]

It is generally thought that the cementicles are formed by calcification of degenerated epithelial remnants in the periodontal membrane. These cementicles could be carried into the gingiva with the continuous eruption of the tooth or as a resut of migration.

The irregularly shaped pink staining bodies (Fig. A and B) appeared to be degenerated connective tissue which had become hyalinized and then calcified. All of the stages from hyalin to calcification could be observed in some of the specimens.

In one instance the calcified body was within the lumen of a vein and could be considered a phlebolith.

SUMMARY AND CONCLUSIONS

This investigation is based on a clinical examination of the gingiva and on a histologic study of gingival biopsies of seventy-six patients, four to seventy years of age. Thirty-six of the patients were under treatment for diabetes mellitus. The remaining forty patients did not suffer from diabetes and the great majority of them were in good physical health.

The clinical examination showed that none of the diabetic patients selected at random were free of gingivitis. There was no correlation between the severity of gingivitis and the control of diabetes.

Histologic Findings:

No characteristic changes for diabetes could be found in the gingiva; however, the following alterations were observed in diabetic patients:

1. Change in surface character from stippled to smooth;
2. Lack of hornification;
3. Intranuclear vacuolization in the epithelium;
4. Increase in the intensity of the inflammation;
5. Change in the structural appearance of connective tissue;
6. Increased presence of calcified "foreign bodies."

Most of the observations were related to the severity of gingivitis. Some of the changes appeared to be accentuated in the diabetic patients and may represent an increased tissue reaction to local irritation.

REFERENCES

1. Aleman, I., and Uleia, I.: New ideas on the oral lesions of diabetes. Rev. de stomatol., **37**:595, 1936.
2. Badanes, B. B.: Diabetes acidosis and significance of acid mouth. Dental Cosmos, **75**: 475, 1933.
3. Bell, E. T.: A text-book of pathology. Lea & Febiger, 45th Ed., Philadelphia, 1941.
4. Burkett, L.: Oral Medicine. J. B. Lippincott Co., Philadelphia, 1946.
5. Gersh, I.: Manual midwest Seminar Dental Medicine. 1948 (Unpublished).
6. Glickman, I.: The periodontal structures in experimental diabetes. N. Y. J. Dent., **16**:226, June 1946.
7. Gray, H.: Am. J. Med. Sci., **168**:35, 1924.
8. Harrow, B.: Textbook of biochemistry. W. B. Saunders Co., 3rd Ed., Philadelphia, 1946.
9. Hirschfeld, I.: Periodontal symptoms associated with diabetes. J. Periodont., **5**:37, January 1934.
10. Hirschfeld, I.: Periodontal symptoms associated with diabetes. Mil. Surgeon, **85**:489, December 1939.
11. Joslin, E.: The treatment of diabetes mellitus. Lea and Febiger, 8th Ed., Philadelphia, 1946.
12. Kronfeld, R.: The biology of cementum. J.A.D.A., **28**:1451, 1938.
13. Lovestedt, S., Austin, L.: Periodontoclasia in diabetes mellitus. J.A.D.A., **30**:273, February 1943.

14. Marble, A.: Diabetes mellitus. Its relation to oral pathosis. J. Periodont., **18**:4, 151, October 1947.
15. Massler, M., Schour, I.: The P.M.A. index in 10,000 persons. In press.
16. McCarthy, Lee: Histopathology of skin diseases. C. V. Mosby Co., 1931.
17. Menkin, V.: Biochemical factors in inflammation and diabetes mellitus. Arch. Path., **34**: 182, 1942.
18. Menkin, V.: Carbohydrate metabolism, local acidosis and the cytological picture in inflammation. Am. J. Path., **13**:1, 25-43, January 1937.
19. Orban, B.: Gingival inclusions. J. Periodont., **16**:16, 1945.
20. Orban, B.: Clinical and histological study of the surface characteristics of the gingiva. Oral Surg., Oral Med., Oral Path., **1**:9, 827-841, September 1948.
21. Ray, H., Orban, B.: Deep necrotic foci in the gingiva. J. Periodont., **19**:3, 91, July 1948.
22. Richardson, K. C., Young, F. C.: Histology of diabetes induced in dogs by injection of anterior pituitary extracts. Lancet, **234**:1098, 1938.
23. Rutledge, C. E.: Oral and roentgenographic aspects of teeth and jaws of juvenile diabetes. J.A.D.A., **27**:1740, November 1940.
24. Schour, I., Massler, M.: Endocrines in dentistry. J.A.D.A., **30**:943, 1943.
25. Simon, F.: The glucose content of human saliva. Dental Cosmos, **68**:622, July 1926.
26. Smith, Lawrence W., Gault, Edwin: Essentials of pathology. Blakiston Co., 3rd Ed., Philadelphia.
27. Ziskin, D. E.: Diabetes in relation to certain oral and systemic problems. Part I. J. D. Res., **23**:5, 317, October 1944.
28. Ziskin, D. E.: Diabetes in relation to certain oral and systemic problems. Part II. Am. J. Ortho., **30**:12, 758, December 1944.

A Case of Acute Gingivitis Apparently Caused by Administration of Dicumarol

By JESSE L. WILLIAMS, D.D.S.
Jacksonville, Fla.

DICUMAROL has been widely used as an anticoagulant in cases of venous thrombosis and in patients who have had pulmonary emboli. The substance is responsible for the "sweet clover disease" of cattle and lowers prothrombin level of blood plasma.

L; — A sixty-three year old male dentist entered the hospital on October 6th, 1949, complaining of a noticeable pressure in the chest. After some ten days, where dicumarol was given daily, the patient noticed a tight uncomfortable sensation in the interdental papillae and also tenderness on masticating food. On examination the marginal gingivae and interdental papillae were congested, edematous and extremely red in color. A slight bleeding was in evidence on gentle massage. Dicumarol was discontinued and, in from two to five days the involved gingival tissues returned to normal and have remained so permanently.

A 6′ film of the chest shows the heart is moderately enlarged, its transverse diameter being 15.7 cm., as compared to the predicted normal of 12.9, indicating about a 22% deviation above normal. The aorta is considered within normal limits. The lungs are clear.

Laboratory reports showed that when dicumarol was first given the prothrombin time was 14 seconds (100% of normal). The next day it was 16 seconds (90% of normal), and at the end of one week it was 20 seconds (44% of normal). On the eighth day it reached 33 seconds (18% of normal), and then stayed below 27% of normal until the drug was discontinued.

Editor's Note: This finding is to be expected occasionally, but is by no means a constant occurrence in patients who receive dicumarol.

Editorials

Specialization in Dentistry

IN THE evolution of dentistry it was inevitable that at some point in its history specialization should develop. It could hardly be otherwise in a rapidly growing profession. The time was bound to come when it would be impossible for anyone to successfully meet *all* the requirements of modern dentistry. And when in the natural course of events that time arrived, there was only one way to meet it, namely, by specialization.

The same thing occurred in medicine, and again the answer was specialization. The family physician cannot and does not undertake the treatment of all his patients. He depends, and rightly so, upon the cooperation of many specialists. Without such cooperation it would be manifestly impossible for him to render the service required. The general practitioner may not like this, but the fact that he finds such cooperation necessary, proves that specialists are needed.

To a less degree this is also true of the family dentist. He cannot if he would, meet all the requirements of his patients. Modern dental practice has become too complex, hence his dependence also on the specialist.

It was out of this situation that specialization in both medicine and dentistry had their birth. In neither case would this have happened had the general practitioner been able to keep pace with a growing profession and meet the demands made upon him by the public.

There are those in both medicine and dentistry who feel some apprehension over this drift toward specialization.* It is urged that the presence of specialties in dentistry may cause educators to neglect the teaching of certain subjects in the dental schools; that they tend to reduce unity in the profession; that there is danger of over-specialization with the tendency of the general practitioner to limit his practice along the lines of convenience rather than in accord with the health needs of the patient; that "the majority of dentists are qualified by aptitude and inclination to practice the entire field of dentistry," etc. Unfortunately, there are no unmixed blessings, (not even in specialization), and probably never will be as long as human beings are human.

Meanwhile specialization continues to go forward. Medicine at present has fifteen specialty boards and dentistry five. Each of the latter conforms to the rules set up by the Council of Dental Education and has its approval. These boards have been organized for the purpose of raising the standards of its specialty group and to examine the qualifications of those candidates who apply for certification. This is done to insure the public as far as possible against incompetence.

---

*Wright, Walter H.—Specialization in Dentistry.
Robinson, J. Ben—The Place Specialization Should Occupy in Dental Practice. The New York Journal of Dentistry, August-September 1949, pp 275-283.

Laws have been enacted in six states governing the activities of specialists in dentistry in those states. By some this is regarded as a mistake. It is felt that dentistry in this respect might better follow the example of medicine by placing control in the specialty boards themselves where it logically belongs. It is urged that since license to practice dentistry carries with it the right to practice it in all its branches, it is unwise to enact laws, to restrict that right as is done by certain specialty laws.

Some specialty boards and at least two states insist that the practitioner shall limit his practice to his specialty. Since it is difficult in some specialties to define its boundaries their diplomas are certified "as expert" in their chosen field. It is believed that the chief concern of the specialty board should be the competence of the candidate to render the service required of him, rather than insistence upon some arbitrary restrictions that may or may not be in the interest of the public. This would seem to be a reasonable attitude.

That specialties are here to stay there can be no doubt. They are the natural out-growth of the multiplication of skills required in modern dental practice. Through these specialties dentistry is able to render a far more comprehensive service than would be possible without them. There would seem to be no cause for fear from "an epidemic of specialization fever."

A. H. M.

Specialization: A Warning

AND THEY said one to another: Go to, let us build an institution, a tower whose top may reach unto heaven; and let us establish ourselves and make us a name, lest we be scattered upon the face of the earth. And it came to pass that the people prospered and the structure grew, and the workers became as numerous as the grass of the field.

But behold there rose up specialists; some there were who were *orthodontists*, some *prosthodontists*, some *exodontists*; others there were who were *histologists*, *radiologists*, *pedodontists*, and *periodontists*. Then did each group cleave unto itself and forsake the others, and the work on the tower did begin to suffer.

And lo, confusion grew and multiplied throughout the land. Then arose a cry from the workers: Let ivory be brought for the completion of the tower. And it came to pass that the specialists did multiply and did form themselves into lesser groups of specialized specialists until all progress ceased, for there was a strange confounding of language and they understood not one another's speech.

Then were the *dentists* without unity; then did their common heritage slip from them, and they became scattered like leaves over the face of the earth.

And the tower fell into ruin and decay. Therefore is the name of it called Babel, because the speech of the people had become as divers strange tongues and as the clashing of cymbals.*

*Modified from an article by James I. Brown. Freshman English and General Education. J. Higher Education **21**:17 (Jan.) 1950. Italicized words changed by M. K. H.

Bio-mechanics of Periodontal Disease

ERNEST R. GRANGER, *Mt. Vernon, N. Y.*

DENTISTRY in general (and periodontia in particular) stands today acutely in need of a practical, rational philosophy, based upon a biologic concept of the human mouth; a bio-mechanical basis for dental practice. These are times of rapidly changing philosophies and values in all human relationships, and dentistry dare not be an exception. This is not some abstract academic problem, but a practical understanding which a dentist must possess if he would avoid confusion and failure.

Philosophy is an understanding of the causes leading to any given effect. It is not here concerned with the technique for performing any given operation, but rather with what operations are required to make this a healing profession; with an understanding of the relation between the mechanical procedures of dentistry and the biologic phenomena occurring in the tissues of the jaws, to the end that the dentist may in a very real sense be a physician to the mouth, instead of a repairman dealing with a lot of static, unrelated pieces of inert matter.

The lack of such a philosophy is the most acute, far reaching defect in dental practice today. It casts its lengthening shadow over the entire future of dental practice, colors all of the future planning for dentistry, and tinges our relations with allied professions. Because of its lack dental thinking wanders aimlessly from one technique to another, from one material to another, in an endless search for a cure-all, without ever conceiving the real problem. Technique and materials are the tools of dentistry, they are useful only as a means of fulfilling our biologic objectives.

Scientific discussions and research in dentistry concern themselves very largely with dental caries. Clinics and papers on restorative dentistry concern themselves largely with the problems associated with the "fit" of dental appliances and caries; dentists in actual practice think and act largely in terms of fit. But from the standpoint of preventing loss of teeth from caries and lack of fit, among these people who do go to the dentist, our knowledge today is adequate. If the problem of maintaining healthy mouths were simply a question of dealing with dental caries and proper fitting replacements, we could prevent the loss of most teeth as far as their health is affected by these factors alone. Failures in this respect are largely due to carelessness, or failure on the part of the operator to use the means we have at hand. If tomorrow we were suddenly to discover the cause and prevention of dental caries, we would still have to deal with diseased mouths and loss of teeth, but the majority of the profession would be at a loss to know what to do.

The fact remains that even with dental caries as widespread as it is today, among the public at large four teeth are lost from periodontal disease to every one lost from caries. But among those patients who receive regular dental care, ten teeth are lost from periodontal disease to every one lost from caries. From comparison of these figures we might draw two inferences. Either filling the cavities has in some way created more periodontal disease, or by preventing the early loss of teeth from caries, the dentist has merely preserved them for the subsequent attack of periodontitis. But in either case, it is readily apparent that the real problem of the practicing dentist is not dental caries, but the preservation of the supporting structures to prevent the inroads of periodontal disease. And those measures which, taken in time could prevent the breakdown, are the same measures which the periodontist must take if he would bring about a cure

Presented at the Thirty-Third annual meeting of the American Academy of Periodontology, October 13, 1949, in San Francisco, California.

for a sick mouth, so that nature's own metabolic processes can maintain that mouth in a state of health.

Fundamentally of course we are dependent upon nature's own metabolic processes to maintain the health of the mouth. But there is nevertheless a direct and definite relation between the mechanical procedures of dentistry and the metabolic activity of the adjacent structures. So first let us try to view dentistry as the treatment of a dynamic living mouth.

Bacteria are not the cause of dental disease. If they were, all of us would be sick all of the time. Bacteria produce the symptoms which we designate as various diseases. In the mouth bacteria produce the symptoms which we call periodontal disease. By symptomatic treatment, elimination of the local irritating factors, and the use of various antibacterial measures, and physical therapy with the tooth brush and similar devices, we can produce the appearance of health in the tissues. But the loss of bone and formation of the pocket were what brought about the local irritating factors. Unless we deal with the factors which permitted the formation of the pocket in the first place, even though we combat the symptoms of infection and maintain the appearance of health, the teeth will be lost just the same. That is what we mean by treatment of the whole mouth.

CLUE TO DENTAL PROBLEM

Fundamentally, the health of any tissue depends upon the ability of the nerve and blood supply to fulfill its metabolic requirements. The sympathetic and para-sympathetic nervous systems constituting together the autonomic nervous system, bring about the breaking down, repair and growth of these tissues which together composes its metabolism. The autonomic nervous system is in turn controlled by the glands of internal secretion. Witness for example the excess growth of tissue brought about by overstimulation of the para-sympathetic nervous system by excessive thyroid activity. We know comparatively little about the actual mechanism, but we do know that in what we call a healthy individual the glands of internal secretion balance the autonomic nervous system and determine the utilization of the blood supply by the cells which compose the tissues. Herein lies the clue to ageing of the tissues and the degenerative diseases. It has for example, been possible experimentally to halt the progress of early periodontal disease by stimulation of the para-sympathetic nervous system, but it has not been possible in advanced cases. Here then is the clue to our dental problem: the link which connects the technical procedures of dentistry to the health of the mouth. If we can once understand that clearly, the veil is lifted and many of the apparent mysteries of dentistry dissolve into thin air. While we know comparatively little about the mechanism of the endocrine glands, fortunately as dentists we do not need to know. That is not directly our problem. Even if we did know we could not by that means alone produce a healthy mouth. It is of interest to us only insofar as it helps us to understand our dental problems. We do know that basically the health of any tissue is determined by the ability of the nerve and blood supply to fulfill its metabolic requirements. But here is the important corollary. The metabolic requirements of any tissue are determined by its functional demands. Therein lies the crux of our whole dental problem. The ability of the body to maintain any tissue in a state of health is related to function. Here is the answer to occupational disease, and physical therapy, and the basic reason for existence of any specialty of medicine. It is the peculiarities of the functional problems of dentistry which set it apart from the other specialties. It is also the reason why periodontia is the practice of dentistry.

Let me digress here for just a moment to explain that a little more fully. We know comparatively little about the workings of the autonomic nervous system, but recent investigation in poliomyelitis have given us a clue to the connection between traumatogenic occlusion and breakdown of the supporting structures of the teeth. The autonomic nervous system consists of the sympathetic and para-sympathetic nerves. The sympathetic nervous system controls the

destructive processes involved in utilization of the tissues. The para-sympathetic nervous system controls the growth and repair of these same tissues. The functioning of these two sets of nerves is under the control of the glands of internal secretion. It has been possible experimentally to cause local growth of tissue by continuous local stimulation of the para-sympathetic nerve to that part. Today we do know that the health of any tissue is directly related to the ability of the nerve and blood supply to meet its metabolic requirements. Of course, the blood must supply the materials necessary to that metabolism which is where diet enters the picture, but the ability of the tissues to take and use that depends upon the autonomic nervous system.

But the dentally important corollary to that is the fact that the metabolic requirements of the tissue are directly related to its functional demands. We have then three factors which enter into the problem, and on this basis we can readily explain some of the apparent mysteries and contradictions of dental treatment. It has been possible to arrest the progress of some cases of early periodontal disease by stimulation of the para-sympathetic nervous system, yet more advanced cases have failed entirely. That is because in the advanced cases loss of bone has increased the functional demands upon the remaining bone beyond the ability of the body to satisfy its metabolic requirements no matter how much stimulated.

Dentistry, and particularly the periodontal problem, presents an amazing array of apparent contradictions. But they are only apparent. If we can get a clear overall picture, we will discover that they are merely different facets of the same problem. In recent years dentists have lined themselves up into two groups who seek to explain the puzzling failures of dentistry. On one side we have those who believe it is a problem of external factors, and on the other side those who believe it is intrinsic to the mouth itself. In both of these groups we find many disagreements and opposing ideas. Briefly, in the first group are those who place their faith in vitamins, then others who go further and believe it is a nutritional problem. Still others, particularly among the periodontists, dismiss it as some hazy general disturbance. More recently, the study of dental medicine has sought to tie up various medical diseases as the villain of the piece, even invading the field of psycho-somatics and blood dyscrasias. The second group, those who believe in intrinsic factors, are much more sharply divided in their beliefs. But they do have one thing in common, perhaps without even quite realizing it. They are all attempting in one way or another to treat it as a problem of function.

RELATION OF FUNCTION

Each group can point to some degree of success as proof that it is the right one. And each one can explain away its failures in a like manner. But as a matter of fact, careful and detailed analysis will show that they are all proving substantially the same thing. It is the relation between metabolic ability, and metabolic demands as determined by function, that determines the course of disease. If the functional demands are in excess of metabolic level, the tissue disease must follow. That is the kernel of the whole dental problem. If we had time, we could take every one of these apparent contradictions, and discover that they all possess that one common basis. That is the basis of various occupational diseases, and in a very real sense, dental disease is an occupational disease. Where a large number of people are engaged in an occupation which requires constant use of some part of the body, some of those people will develop in varying degrees an occupational disease which is peculiar to that trade. The reason of course is that the metabolic requirement created by the functional demands upon that particular part are beyond the metabolic ability of some of the people concerned. The varying degrees of metabolic deficiency would determine the varying degrees of the occupational disease. The changes in metabolic activity with age would determine how long he could maintain the functional demand without disease. For the same reason that all people engaged in the same occupation do not develop the same occu-

pational disease, all mouths in the same function do not develop the same periodontal disturbances.

Here we have an explanation for many puzzling things we have all seen. Mouths in very bad malfunction which do not break down, others which with much less malfunction break down badly; why the most careful tooth grinding in the world can never be wholly successful, yet various methods produce varying results; the varying results of dietary treatment; those hazy systemic factors; why fairly simple restorations of function sometime produce a favorable result; the variation in results we can obtain with even the best of function.

Every dentist knows that a very important determining factor is the amount of bone which is left around the tooth. Have you ever stopped to wonder just exactly why that is true? If it is not a problem of function, then what difference does the amount of bone make? There is no other possible explanation. Whether we realize it or not, it has been firmly established beyond any doubt that the problem of maintaining healthy supporting structures is a problem of function. In the sense that it is related to the metabolic problem, the man who seeks relief in external factors is partly correct, but that should be in its proper perspective as a means of treatment.

THREE COURSES OPEN

Theoretically there are three courses open to us. We might use any one or all three. First, we might invade the field of endocrinology and try to produce a more favorable balance in the autonomic nervous system. There are many reasons why that is too dangerous a course to consider as a routine practical measure. Secondly, we could try to build up the metabolic constituents of the blood. That is perfectly practical, within the limits of our present knowledge of what those factors are. Indeed, we must give it all possible consideration. Obviously, anything which affects the health of the entire body will be reflected in the mouth. We cannot deal with dental problems as though they were entirely separated from the general health. But by that same token they enter into our problem only to the extent that they do influence all the tissues. The degree of success we can attain by that means alone is limited by the amount which metabolic ability falls below functional demands. And they would be useful to us only to the extent that they are required for the health of the whole body. But what about that vast majority of people whose metabolism is just not equal to the excessive functional demands of the mouth? Here we can get into a vast complication of what we conceive to be health, or is there such a thing as a perfectly healthy person, which is very likely to throw a smoke screen over the whole picture. If we can look through to the heart of the problem we can see that we have but one logical approach. Our constant aim; the basic consideration upon which all our technical procedures must be predicated, must be to create and maintain the best possible function. We know that the more perfect the function, the less the metabolic requirement. In spite of our best efforts, metabolic ability is bound to fall as age progresses. But the more perfect the function, the lower it can fall and the longer it will be before functional demands exceed metabolism.

There is a great deal more that should be said on this subject, particularly as regards the relation of functional disease to growth physiology, maintenance physiology, age changes, and a host of other aspects. We have only scratched the surface. Here we are only attempting to show that there is a connecting link between the mechanical procedures of dentistry and the health of the supporting structures. But that does not answer our problem. On the contrary, it only shows us the reason why it is so important that we learn the correct answer to the real problem. What is the most perfect function? If we can recognize the fact that the health of the mouth is directly related to the function of the mouth, that the better the function, the longer metabolism can maintain it, then surely we must see that our real dental problem does not lie in the field of medicine, but rather in the mechanics of function; that we must first understand what is theoretically

perfect function and then make our compromise approach that as closely as possible within the physical limitations imposed upon us in the hope that nature's metabolic processes will provide the necessary tolerance. The answer to that is infinitely more difficult to explain, requiring literally years of patient study if we would have a really complete picture.

The most difficult problem we face in any discussion of dentistry as a treatment for a sick mouth, is the lack of a general concept by dentists as a group of the relation of oral physiology and anatomy to our actual practices. Anatomy is usually shrugged off as something we learn in college and then promptly forget, and besides what in the world has it to do with patching holes and bridging spaces. Upon frequent occasion we hear from the lecture platform high sounding phrases about the physiologic factors of the mouth. It is always some nebulous intangible thing which is just above us but we all derive a glow of satisfaction and pat ourselves upon the back that we are members of a learned healing profession. But when Mrs. Jones comes into the office as a patient we forget all about it for it has no practical application to our routine daily work.

But anatomy is the dynamic form and relation of parts which makes physiology possible. These two must always be considered in their inseparable relation to each other and to the practical problems of routine daily practice.

ORAL PHYSIOLOGY

Unfortunately, not only do dentists generally not understand the know how of oral physiology, most of them do not even understand what we mean by the term itself. When dentists think of biologic factors, they generally start talking in terms of the general chemistry of the body as it applies to all of the healing arts. They think about such things as blood counts, urinalysis, calcium metabolism and similar medical problems. It is quite true that all these factors can and do exert an influence upon the health of the mouth, particularly as regards diet and the vitaminoses.

We cannot ignore these factors. The mouth is a part of the body, and obviously anything which affects the health of the whole organism will be reflected in the mouth. By that same token, the degree to which those factors can enter into the health of the mouth is subject to the same influences that determine how much they will affect any other organ of the body. And that is where our problem differs from the general medical problem, there begins the biologic problem which is peculiar to the practice of dentistry and dentistry alone. Those dentists who merely try to ap the physician with drugs and diets and vitamins can never bring about a permanently successful result, because these things alone are not enough. The degree to which these can help is determined by the physiology of the mouth. It is the relation between these factors and the mechanics of tissue pathology which constitutes our routine daily dental problem.

We have heard a lot about reconstruction, as though it were something set apart from "ordinary" dentistry. The word reconstruction is indeed a poor choice, for in dentistry it has come to mean many things to many men. If we accept it literally, what we mean is to restore a mouth to its former condition. But if the conditions which prevailed in that mouth have caused the original degeneration, we could hardly wish to restore them. Unfortunately, whether the dentist intends it or not, that is what actually happens in far too many cases. Strictly speaking, any restoration upon a tooth is a reconstruction, but as widely used, the term implies a definite difference between it and "ordinary" dentistry. When we look into a mouth and by simple visual examination determine that that mouth is in need of reconstruction, we, in that simple statement, imply a great many things. We imply that reconstruction is something different from just restoring so many teeth; that it is something better or else we would not do it; that therefore what we conventionally call good dentistry is not so good after all; that by reconstruction what we really mean is to do things which should have been done in the beginning, which have always needed doing; that at long last we

are finally going to treat the whole mouth.

We refer to a "good" filling or a "good" bridge without the slightest regard to the rest of the mouth, but when we reconstruct a mouth we suddenly recognize the importance of relating each restoration to the whole. In our minds we recognize the fact that it is perfectly good practice to do what we conventionally call "good" dentistry until conditions have become bad enough to force us to reconstruct that mouth. But the mere fact we recognize also the need for reconstruction presupposes that in our minds we know the difference. If we admit that reconstruction means something more, that what we really expect to create is an improvement over the conditions which formerly existed in that mouth, then we imply that something is fundamentally wrong with our entire philosophy of dental practice; that we are not being intellectually honest with ourselves. In all truth dentistry, to be completely successful, requires a philosophy *more* than a technique.

REQUISITES OF GOOD DENTISTRY

Reconstruction is, in fact, just good dentistry; conversely, to do good dentistry we must do the same things that we do when we finally face the true facts. It would seem that the requirements of reconstruction are really the requirements of good dentistry. For those factors which we recognize as being the cause of breakdown; the things which we know we must do to successfully reconstruct a mouth, are the very same things which, taken in time could have prevented the degeneration and the need for extensive restoration. Had we considered that same mouth, from the beginning and all through the years of dental treatment (neglect might be a better word) in the same light that we do when we are trying to arrest the destruction that has occurred, we could have created conditions that would make it possible for nature to maintain a healthy functioning mouth. That is the only possible basis upon which dentistry can hope to become a treatment for sick mouths. Drugs and physical therapy can never replace rational therapeutics. When a man says that he does not believe in reconstruction, what he really means is that he does not understand what it is that makes a healthy mouth. We pride ourselves on the wonderful mechanical perfection of modern dentistry, but we must honestly, with an open mind, face the fact that dentistry, as widely practiced, does not cure dental disease, it only maintains comfortably diseased mouths, many of which would not exist if it were not for modern dentistry. Dentistry as a profession does not recognize what dental disease is. We only consider its end results. This may sound harsh and unjust, but if dentistry is ever to stand on its own feet and deserve the recognition it claims as a healing profession we must face this issue. It has been said "that American dentistry shows considerable skill in the construction of parts for the mouth; our technique is the envy of the world. But we are only makers of parts which bear no relation to the whole. The assembly of these parts often results in a ruinous mechanism."

Our ability to save teeth that under less skillful treatment would once have been lost, has resulted in the anomalous condition that in an alarmingly large percentage of cases we are actually doing the mouth more harm than good. For what we are doing is to preserve comfortably diseased teeth which are a slow insidious menace to the health of the patient. Indeed, so accustomed have we become to universally diseased mouths, and so little do we know about them, that like the common cold it has become a part of our daily lives, and not until pathology has become gross and disease well nigh beyond hope does the dentist give it serious consideration. One of the strange reasons for this lies in the fact that dentists themselves do not genuinely appreciate the importance of the mouth as a vital organ of a healthy body. We pay lip service to it, but deep down in our hearts we are not truly conscious of it in our daily work.

Broadly speaking, with respect to the part that teeth play in mouth function we have two opposing schools of thought. On one side we have those who believe that the teeth themselves rule the entire organ, and at the

other extreme, those who believe they exercise no control. I personally believe that the teeth are simply a harmonious part of the machine working in conjunction as equal partners in a cooperative enterprise. That they are neither the masters nor the servants of the temporo-mandibular joint. That they share equal responsibility and equal control. In short, we cannot put whatever teeth we like into a mouth and expect that mouth to adapt itself to them, but neither can we neglect to create tooth forms which will in function exercise that measure of control which is necessary to the proper coordination of all these parts. But the teeth must all share this control equally among themselves and in complete harmony with each other as well as with the rest of the organ.

These functional forces involved in chewing with cusps are the biologic factors which are peculiar to the practice of dentistry and dentistry alone, which set it apart from the other specialties of medicine. They are true biologic factors because they are the functional demand which determine the metabolic requirement, or the ability of the metabolic level, to maintain the tissues of the mouth. They determine whether dental treatment is a restorative remedy, or a foreign body to be thrown off; whether our mechanical devices are biologically sound.

PROBLEM IS BIO-MECHANICAL

Our problem then is Bio-mechanical. Dental treatment by its very nature must be mechanical, but that does not mean that it cannot be Biologic. The surgeon, the oculist, the physical therapist, all employ mechanical means, but always with the biologic objective of restoring and maintaining function. The problem we face is much more intricate than theirs. Certainly we can do no less. All mechanical replacements must in function exert leverage of one type or another on their supporting and opposing structures. Since it is a fact that in most cases by the time treatment can be undertaken some degeneration has taken place, it is all the more important that they be in biologic harmony, so that nature's healing process can come to our aid. We may be able to correct deformities, to supply some of the things which nature has failed to do, but we can never neglect the maintenance physiology of the body as the basic requirement of a healthy mouth. Here then must be our basis of dental practice, if we would create and maintain healthy functioning mouths, and avoid that inevitable set of full artificial dentures. Whatever mechanical procedures are indicated in a particular mouth, they must always be predicated upon the maintenance physiology of that individual. Whatever oral treatment the diagnosis shows necessary to correct in tooth repair or in tooth replacement, or in tooth removals, we must be guided in our prescription by the mechanics of tissue function required to permit that maintenance physiology to operate. Unless the mechanics of tissue pathology receives adequate attention, the degenerative process will continue with ultimate loss of teeth, regardless of local conditions of the soft tissues.

For many years discussions among dentists who have devoted serious study to the mouth have centered around the relation between form and function. On one side one hears arguments to prove that form creates function, and on the other side equally logical arguments that form is the result of function. There is no means of knowing which is correct, and as a matter of fact for the moment from a practical standpoint it is largely an academic question. All of them do agree that there must exist a harmony between form and function, and one clinical fact is firmly established beyond a shadow of a doubt. For the natural metabolic processes of the body to maintain a healthy mouth, the form of the teeth must be harmonious with the functions of the mouth. With that knowledge there is one short sentence to be emphasized. "The practice of dentistry is the science of articulating teeth." When one examines a patient, look at the whole mouth; look at the woods, not the trees. When one sees rampant decay, and chronic gingivitis, and bone destruction, and loosening abutments, and dentures that won't stay put, and tender sore teeth, and obscure facial pains whose etiology does not seem clear, look first at the function of the

whole mouth, not the local condition. Gradually one will begin to get a different view of dentistry, a clear overall picture of dental practice, of which this is the cornerstone, "the practice of dentistry is the science of articulating teeth."

The Problem of Function in Periodontal Treatment

WILLIAM BRANSTAD, D.D.S., *Saint Paul, Minnesota*

THERE has been much complacency among dentists generally with averaging the function of the masticating apparatus. If the upper and lower teeth were capable of meeting in some sort of occlusal contact it has been taken as an indication that the functioning of the mouth was normal and satisfactory. This relationship may be physiologic or pathologic depending on how well it happens to conform to the fixed biologic factors of any particular mouth. These are the hinge axis, mandibular centricity, or centric relation, the condyle paths, and the Bennet movement, which is the bodily side-shift of the mandible and is really a part of the condyle path.

Periodontists, however, have long recognized the importance of the correction of traumatogenic occlusion or malarticulation in the treatment of periodontal disease. Many articles have appeared in the literature suggesting orthodontia, reconstruction, and various tooth grinding procedures, singly or in combination, to correct occlusal disharmonies. Most of the emphasis generally seems to have been placed on selective grinding performed directly in the mouth. Some men in this field now believe that to attempt these corrections of a malarticulation directly in the mouth, without an adequate study and diagnosis of mounted casts, is merely to chase the pathology from one tooth or area to another. Some authors have suggested the mounting of study models on various types of articulating instruments to study the occlusive and articulative relations of the teeth and to determine the necessary corrections.

As Dr. Granger has pointed out elsewhere in this issue of the JOURNAL OF PERIODONTOLOGY any really practical method of establishing physiologic function to promote health of the teeth and their investing structures must be based on a knowledge of just how the mouth does function. This is, therefore, an attempt to describe briefly these biologic factors which we must necessarily understand in order to create and maintain a physiologic articulation.

There have been many theories, hypotheses, and techniques advanced to explain and measure jaw function, and for obtaining functional, balanced or working arrangements of the teeth as the instruments of mastication. There have been scores of articulating devices invented, and many of them used in attempting to obtain proper moving relations of models of the mouth as the basis of articulating teeth. There has been much confusion and many have become discouraged with efforts to actually determine and duplicate the function of the mouth on an articulating instrument. Inability to make teeth with cusps function properly has led to the use of cuspless teeth in both fixed and removable prosthesis.

It is difficult, if not impossible, to describe anything anatomic in exact geometric terms. The entire oral mechanism is designed to function with curved surfaces and on curves.

Presented at the Thirty-third Annual Meeting of the American Academy of Periodontology, San Francisco, California, Oct. 1949.

This is one reason why jaw motion has been so difficult to understand. It emphasizes the logic of an approach which records actual jaw relations, and their detailed study, on an instrument capable of reproducing them. At the present time I believe this is the only practical way of determining what corrections or restorations are actually necessary to enable the mouth to function physiologically. An adequate articulator is to oral diagnosis what the microscope is to the pathologist or bacteriologist, and its real value will be proportional to how well we understand oral physiology, or the basic biologic factors of the mouth that determine how teeth articulate.

While we need to learn more about oral physiology and its basis, jaw movement, we also need to utilize what knowledge is now readily available. We are indebted to the Gnathological Society of California, who, through years of research and study developed the now well established basic knowledge of mandibular movement and physiologic articulation of the teeth. As a result of their work they developed the only really accurate instruments with which it is possible to record and duplicate actual dynamic jaw relations.[1]

When the importance of a physiologic articulation and the basic factors involved are more widely understood and appreciated, it is possible that simpler methods of achieving harmony between jaw and motion and our corrective procedures may be developed.

McCollum has said "Fear of more and newer knowledge is a common attribute of every profession. This fear often becomes an excuse for non-acceptance. Often this fear is founded in the uninitiated upon the results obtained by those who prostitute the principles by attempts to 'Make it easy.' There is no royal road to diagnosis in dentistry. Dentistry is difficult, and the more we know about it the more difficult it becomes. It takes more than the ordinary man or mind to master its difficulties. These difficulties are what set dentistry apart, and the mastery of them could give dentistry its place among the learned professions."

Since dentistry is the science of articulating teeth, it would seem that learning what it takes to articulate teeth correctly should be its first order of business.

Let us then consider each of the fixed components of mandibular movement. The condyle paths of the temporomandibular articulation have definite recordable movements that can be reproduced on a suitable articulator. The Bennet movement, which is the lateral bodily side-shift of the mandible, can also be recorded and reproduced. That the temporomandibular joint is not a loose sloppy mechanism which will conform to any given set of tooth forms and arrangement is attested to by the fact that repeated tracings of the same paths may be superimposed, one over the other, and they will be identical in character.

HINGE AXIS

The hinge axis is that axial center within the heads of the condyles, constant to the mandible, which determines the arc of closure on which the cusps meet in any occluding position of the teeth. It can be accurately located by trial and error with a hinge axis bow rigidly attached to the lower teeth or jaw. For convenience the hinge axis is located with the mandible in centric relation. With its location known it is then possible to orient models of the teeth in their proper relation to the opening and closing axis of the articulator. The cusps can then approach each other on the same arcs as they do in the mouth. The importance of this step cannot be overemphasized.

The procedure of using face bows for the transfer and mounting of casts on an articulator which depends on some arbitrary location of the area of the condyle or the hinge axis has led to difficulty because of its inaccuracy. It is no better than the running of a line from the rim of a wheel to some arbitrary spot on the hub and then drilling an axle hold at that point regardless of its eccentricity. The example has also been used of slightly mislocating the hinges on a door and trying to make it close properly.

Centric relation is the terminal hinge position of the mandible from which the Bennet movement may take place. Centric oc-

clusion should take place as the mandible is closing on the hinge axis in its centric relation. If the teeth do not interdigitate properly in this position they are spoken of as being out of centric or in an eccentric relationship.

Every patient can provide a centric relation but it is the dentist who must provide and correlate a physiologic tooth arrangement to that centric. Many patients who have eccentric intercuspation will maintain that they always close in that position, yet an examination of the worn facets on the teeth would seem to indicate that they close on the hinge axis in centric until forced to slide into some eccentric position by cuspal interference. But when told to open and close his mouth, the patient can and will close repeatedly in any eccentric relation to which he has become habituated. The real importance of centric relation does not lie in the question of whether the patient does or does not close his mouth in centric. That is merely a quick and easy way of detecting an eccentric relation. Centric is the "bracing" position of the condyle as the mandible rotates about it and slides laterally in the Bennet movement on the working bite in lateral excursion. To picture that more clearly, let us briefly outline the steps in jaw movement during mastication.

OUTLINE OF JAW MOVEMENTS

As food is introduced into the mouth the mandible opens downward and forward to grasp it. The teeth close upon it in lateral protrusive and the mandible slides back toward centric. Now the teeth open sufficiently for the tongue and cheeks to set the bolus of food between the teeth. The separated teeth now move into lateral excursion, preparatory to closing upon the food and returning toward centric. As the teeth start through the bolus of food, in lateral excursion, the temporal and masseter muscles contract to provide the necessary force for mastication. The condyle on the working side is now braced against the distal part of the glenoid fosa. The mandible in lateral excursion, is closing on the working side in its terminal hinge position. It continues on through the bolus of food until the teeth meet, still in lateral excursion. If the cusps are properly related to each other, the upper and lower teeth interdigitate and the condyle is not pulled out of its bracing position. Now the mandible, with the teeth together, glides back to centric, the head of the condyle rotates in the fossa, as it executes the Bennet movement, and the cusps pass between each other with a shearing and shredding movement. The glide ends when it reaches centric, the mouth opens and again moves laterally to close in its bracing position and repeat the stroke.

But now let us trace the chewing process if the teeth are "out of centric." As before the teeth grasp the food in lateral protrusive and return. The teeth open and the bolus is carried between the teeth. The mandible moves into lateral excursion with the teeth separated, and the teeth close into the bolus preparatory to shredding it on the return glide toward centric. As the teeth close with the condyle in its distal bracing position under muscular force, one or more cusps meet in premature contact, and prevent the remaining cusps from interdigitating. As the muscular force continues to carry the teeth through the bolus of food, the teeth are forced to slide forward on the premature contact until the cusps do interdigitate. The condyle is pulled forward so that it is no longer braced against the fossa. Now the entire muscular force is braced against the cusps which are holding the mandible forward. This force is transmitted as lateral pressure against the bone supporting these cusps, instead of the vertical thrust in normal relationship. As the cusps glide past each other and the mandible attempts to return to centric, it exerts constant pathological pressure upon the bone. During the entire time that the heaviest muscular force is being exerted on the working bite, it is all transmitted as a lateral thrust against the supporting bone of the premature cusps. In course of time that bone is destroyed by the excessive functional demands, other cusps come into premature contact and the process continues on other teeth. Frequently it happens that at different stages of the masticat-

ing movement different cusps are in premature contact at different times, but at each stage the cusp that is premature is forced to take the entire bracing of the mandible. The varying degrees and direction of lateral pressure produce the varying pattern of bone destruction.

The combination of rotation and gliding medially, of the condyle on the working side, is the Bennet movement. Since it is the power movement, it is the most important of the mandibular synergy. Because the Bennet movement is the power movement and takes place with the condyle braced against the fossa, it is the reason why centric relation is of fundamental importance, regardless of what method may be employed to achieve it. To record the condyle path without the Bennet movement is worse than useless. It is actually misleading in diagnosis, and in treatment is capable of more potential harm than good. Conversely, to restore "centric relation" medially, without consideration for the Bennet movement, will requently make a bad matter worse, and actually hasten the destructive process.

On the balancing side the effects of eccentric relation are not so readily discernible. Here the condyle is down and forward on the condyle path, as the teeth close upon the bolus, in lateral excursion on the other side. As the working side glides medially, the condyle on the balancing side returns along its path toward centric, until it meets the premature contact at the point of eccentric relation. There may of course be other points of premature contact as it is making the return stroke. As it is stopped by the interdigitation of the cusps at the point of eccentric relation, it attempts to return all the way to centric, to complete the rotation on the working side. All of the thrust now is against the cusp in premature contact on the balancing side, transmitted as lateral pressure against the bone.

Occlusion has been discussed so much that definition seems unnecessary but it should be clearly understood in its relation to articulation. Articulation is the dynamic anatomical relation of the teeth in every possible contacting position, or the change from one occlusion to another with the teeth in contact. Occlusion is the contact relation of the upper and lower teeth at any given stage of articulation.

Recording jaw motion is not a quick and easy way of determining physiologic tooth form and alignment, but it is at present the only method of achieving this ideal. Instead of guessing or using averages, having certain definite records from the head to work with really simplifies determining what the proper correction or restoration should be.

We have been concerned here with the fixed biologic factors of the mouth, the hinge axis, centric relation, the condyle paths, and Bennet movement. There are other factors which are not anatomically fixed such as the plane of occlusion, curve of Spee, and anterior guidance which influence the form and character of the cusps and their articulation. These variable factors are to a degree under the control of the operator.

The cusps, sulci, and fossae of the teeth that will function properly in any given mouth are the result of these fixed and variable factors. Because of the infinite variety of possible combinations of these factors, cusp height and form varies widely between individuals. Under any given fixed set of these factors there is only one set of cusps that can function in harmony with jaw movement.

Inasmuch as one must create cusps which will function in harmony with jaw motion, it tends to indicate that merely grinding off the interferences can never be wholly successful because it fails to restore the areas of occlusal contact essential to a physiologic articulation.

BIBLIOGRAPHY

1. McCollum, B. B., and Stuart, Charles E., A Research Report. Dental Items of Interest, June, July, August, September, October, November, December 1939 and January and February 1940.

In Our Opinion

More About the Occlusal Factor in Periodontal Disease

Initial Statement

By Harold J. Leonard, D.D.S., B.A., *New York, N. Y.*

IN THE "In Our Opinion" Department of the 1946 Journal (17:80-91, Apr. 1946) and in a separate article (id. 14:12-24, Jan. 1943) this subject was discussed and the following causes enumerated for occlusal stresses which tend to injure the periodontal tissues and thus predispose to periodontal disease:

1. Loss or absence of one or more teeth or contacts resulting in shifting of other teeth, and failure in mesiodental support.
2. Uneven wear resulting in unworn cusps which interfere.
3. Orthodontic defects in which teeth are erupted in unstable positions, or are so placed as to interfere with free mandibular movements.
4. Injurious mouth habits such as habitual clenching of teeth; pushing against teeth with tongue, walking with heavy pipe in mouth, thread biting, and other occupational pressures.
5. Periodontal swellings due to inflammations or tumors that push the teeth into positions of occlusal interference.
6. Improperly made fillings, bridges and dentures which create occlusal interference or through faulty clasps cause lateral thrusts.

At the October 1949 Meeting of the Academy Ernest Granger of Mt. Vernon, N. Y., read a paper in which another cause was presented. This is a condition in which maximum intercuspation does not coincide with the true centric position. The true centric position has been defined by D. W. McLean of Los Angeles (J.A.D.A. 29:1204, July 1942) as the most retruded position of the mandible from which functional movements may be made with ease and comfort. It is a skull-mandible relationship to which the teeth normally adjust at the time of eruption. According to him, it does not change position even though the position of maximum intercuspation does not coincide with it and the teeth habitually close in an eccentric position. When maximum intercuspation does not coincide with true centric position there are strains and stresses on the teeth. The muscular effort is to close in the true centric position but since there is cuspal interference the jaw slides on the inclined planes of the interfering cusps into the position of maximum intercuspation. Eccentric intercuspation may be forward of true centric position or to one side. Evidence of a forward position is periodontal destruction on the posterior sides of upper molar teeth and on the mesial sides of the lower.

McLean also speaks of centric interference in which maximum intercuspation coincides with true centric position but in which one or more cusps interfere with

the jaw in the act of closing into that position. Such a condition, he says, can be entirely corrected by selective grinding of the interfering cusps.

Eccentric intercuspation, he says, can be corrected only by orthodontic treatment, by occlusal reconstruction, that is, the rebuilding of all occlusal surfaces of the teeth with hard cast metal veneers, or by extraction and replacement with dentures. (McLean J.A.D.A. 23:928-938 June 1939).

Granger's paper and clinic were a demonstration of the destructive effects on the periodontal structures of eccentric intercuspation, methods for diagnosis and a demonstration of occlusal reconstruction and the benefits therof. Diagnosis and treatment are based on an articulator and facebow of B. B. McCollum of Los Angeles (D. Items of Int. 61:522, 642, 724, 852, 943, 1939) by which it is believed the relationships of jaw to skull and the teeth to each other can be exactly reproduced in all movements. Granger and a few other disciples of McCollum are practically limiting their practices to the occlusal reconstruction of cases of eccentric intercuspation in many of which periodontal breakdown is well advanced. Needless to say such a treatment is very costly and a dentist limiting himself to it can treat only ten to fifteen cases a year.

Much as one must admire the technical excellence of such workers as McCollum and Granger, and must believe that they have carried the subject of occlusal restoration to a mechanical ideal, one must pause before becoming too enthusiastic, to consider practical aspects. It is certainly true that many persons have maximum intercuspation in an eccentric position. It is also undoubtedly true that this condition puts stresses on the teeth often leading to periodontal breakdown, but is it true that such cases can be successfully treated and teeth saved in adults only by means of such elaborate occlusal reconstruction as these men imply? In other words are persons so afflicted and who can not afford the time and money for such treatment necessarily doomed to lose their teeth? It has not been my experience that such is the case.

The structures of the body are so designed as to be capable of carrying many times the load ordinarily carried. All that is required is that these structures be in excellent health and that the load be increased gradually so that strength can be built up as required. It is not necessary to have perfection in articulation of teeth in order to maintain health. Providing the tissues are kept healthy from the standpoint of sanitation, nutrition and blood supply it is often amazing how much potentially injurious stress they will stand without periodontal breakdown. Of course individuals differ in their body chemistry and these differences are reflected in the nutrition and resistance to disease of the periodontal tissues. In some cases it seems impossible to build up sufficient health to withstand even mild mechanical irritation. Such cases are fortunately rare. With these few exceptions it has been possible in my experience to put periodontal tissues in health and so maintain them without any such elaborate system of building ideal articulation as advocated by Granger.

This does not mean that great care, effort and ingenuity can be avoided in diagnosing the occlusal faults contributing to breakdown and in treating them

so as to relieve or offset them. It is first necessary to recognize by the nature of the occlusion or by the clinical and radiographic signs and symptoms and by appropriate tests that an actual or potentially injurious occlusal disharmony exists. Second, it is necessary to determine its cause, and thirdly, it is necessary to design a treatment that will offset or reduce the injurious stresses to the point where they can be tolerated by tissues kept in a state of vigorous health by the usual periodontal procedures.

In most cases the interferences in occlusion can be corrected by grinding and reshaping interfering cusps and corners according to principles laid down by McLean (id. 1939) Schuyler (J.A.D.A. 22:1193-1202 July 1935) S. C. Miller et al. (Textbook of Periodontia 1944) H. J. Leonard (id. 1943) and others. In some cases various combinations of grinding with crowns and bridges, occlusal veneer inlays, partial dentures and orthodontics may be needed. Increase in vertical dimension of mild degree is often essential for the purpose. Much care and effort are necessary to bring many teeth into function in masticatory movements, to restore the normal curves of the occlusal surfaces and to offset lateral thrusts in the teeth, especially mesial-distal thrusts.

I am convinced that a large proportion of periodontia cases need some form of occlusal adjustment in addition to the usual periodontal treatment if the teeth are to be saved for the lifetime in health. For this purpose the dentist must have a mastery of the principles of occlusion and of the means of correction, including the movement of teeth by orthodontic means. With very few exceptions this can be done sufficiently adequately by the means generally taught in the best dental schools and available to the competent dentist. I have seen exceedingly few cases that did not respond well to such treatments without resorting to such extremely elaborate technic and mechanical perfection as presented by McCollum and Granger. We should keep dentistry as simple and inexpensive as possible and still accomplish our purpose of maintaining the teeth in maximum health, function and beauty.

Discussion

PAUL J. BOYENS, D.D.S.
San Francisco, Calif.

The use of reconstruction work in cases having periodontal diseases or which are susceptible to disease, presents problems of major proportions. That is why resistance forces and directions of these forces, should be studied extensively.

With a certain amount of bone loss, the leverage on alveolar bone is already increased and any lengthening of the crown would further increase this extra alveolar leverage.

If cusps are so carved that they will give the greatest masticating efficiency, then the lateral stresses may be increased enormously. To put young cusps on old teeth can be disastrous.

In redistribution of occlusal forces and changing direction of these forces, a basic

mechanical principal must be remembered; and that is to give such clearances of the occlusal surfaces as to give the mandible the greatest antero-posterior movements. In these movements the posterior support each other.

The anteriors can be shortened or have excessive stresses avoided by other means, such as increasing the curve of Spee.

When anterior posterior movements are restricted, the mandibular biting stresses become lateral, which are more traumatic because the neighboring teeth do not necessarily share the stresses.

This is no criticism of the value of reconstruction work. But, where occlusal forces may become pathogenic, good judgment must be used.

J. S. FRIEDLANDER, D.D.S.
New York, N. Y.

The observation that maximum intercuspation which is incoordinated with true centric position is an etiologic factor in periodontal disease is not new. Clyde Schuyler and Louis Alexander Cohn and many others have noted it long before. The treatment of this condition need be no different from that of other occlusal irregularities of the teeth, the supporting structures of which are weakened.

The choice and extent of the prescription should be primarily concerned with the cause and the degree of the impairment of the periodontium.

Pediodontists know that many such conditions are reversible by routine periodontal treatment. Some conditions, however, require supportive measures to restore and maintain these tissues in health. Often this need be no more involved than reorienting the occlusion by grinding.

Following the treatment, if the survival of the teeth is doubtful, I would not hesitate to prescribe occlusal reconstruction by resurfacing as many teeth as would be necessary to maintain them in health. It is of commanding importance that the prescription fit the disease.

With this thought in mind I do not think it good teaching philosophy to complicate the subject by considerations of expense involved in restoring the mouth to good health. This may better be explored in the teaching of administrative practices.

I do not agree that McCollum and Granger have carried the subject of occlusal restoration to a mechanical ideal. Their philosophy of fabricating cusp anatomy from the tracings of mandibular movement on a free plane is not sound.

1. These tracings may reproduce existing anomalies of temporomandibular joint relationships.

2. The steep cusps which these tracings often dictate and the broad occlusal surfaces which result from attempting to balance asymetrical jaws often introduce leverages which may aggravate existing impaired periodontal membranes and supporting bone.

Biologic and mechanical research and clinical experience have proven that the supporting structures of the teeth tolerate better, those stresses which result from the vertical application of force to the teeth. This ideal of occlusion may be achieved by judicious modification of cusp height and width and when necessary, by splinting teeth with weakened support to teeth better fortified.

ERNEST R. GRANGER, D.D.S.
Mount Vernon, N. Y.

Since your Initial Statement revolves around some of the material I presented at your past meeting, I beg your indulgence to comment on it as briefly as possible and still try to correct what I feel is an erroneous concept of what I tried to say. To adequately answer the questions you raise would require more space than your entire issue.

Eccentric relation is one of the most powerful destructive forces in malfunction. Simple logic would seem to dictate that any attempt to treat the functional aspect of periodontoclasia without restoration of centric relation is merely temporizing with half way measures, slowing the rate of destruction and postponing the evil day without

ever treating the cause. Is that rational therapy? As you point out yourself in the Initial statement, "It is also undoubtedly true that this condition (eccentric relation) puts stresses on the teeth often leading to periodontal breakdown." A little further on you state, "this does not mean that great care . . . can be avoided . . . in treating them . . . (eccentric relations) so as to relieve or offset them . . . it is necessary to design a treatment that will offset or reduce the injurious stresses to the point where they can be tolerated." Is not that exactly the same thing as we are doing, or where would you draw the dividing line on how much treatment the patient warrants?

At another place you point out that "the structures are capable of carrying many times the load" . . . "all that is required is that these structures be in perfect health. I agree. But we do not treat mouths which are in good health. I have repeatedly pointed out that the indication for this treatment is that the mouth is breaking down as a result of the inability of metabolism to sustain the malfunction. As you point out, under such circumstances our object is to determine and treat all the malfunction, of which eccentric relation is a part. That involves two factors, a concept of oral physiology as a basis for understanding malfunction, and the ability to recognize the signs of breakdown before it has become an acute problem.

I gain an overall impression from your statement that you do recognize the importance of and need for treating eccentric relation, but you question the need for a particular method, not on the grounds of a better approach, but solely as a matter of economic expediency. That we really should explore much more thoroughly. I have always stressed the fact, and did so at San Francisco, that I am not primarily interested in what method is used to achieve the result, but only with what we must do to create a healthy functioning mouth. I am all for simplification, indeed have worked hard to contribute to it, provided it is not brought about by simply avoiding our obligations to treat the mouth. And strangely enough, our present methods, in spite of their apparent complexity, do greatly simplify the problem over what it was not so many years ago. The more complex the instruments and the more they will do for me that I do not have to do in the mouth with my own energy, the simpler becomes my problem of treating people. I could not accomplish the same treatment for as many people with so called simpler methods because then I would have to do a lot of things at the chair which I can now do in the laboratory. We figuratively have the patient on the laboratory bench. But remember, I said I could not achieve the same result. We could of course use simpler methods and sacrifice results, but I am afraid I cannot go along with that compromise. If I had a cancer for which there was a known but complicated cure, I would hate to be treated by a physician who used simpler methods just because they were cheaper. Carrying that to its logical conclusion, the simplest and cheapest treatment for periodontitis would be to extract all the teeth and make full dentures. Again, where would one draw the line? At the patient's pocketbook?

That does not mean that I can ignore the economic problem. It is ever present with us. Because economic considerations are such an everyday problem of dental practice, we are all inclined to get mixed up in our thinking about dental treatment. The vast majority of clinicians, perhaps unconsciously, approach dental problems more from the practical economic angle than the scientific problems of mouth health. It has always seemed to me that is putting the cart before the horse, and has greatly retarded dental progress. That may influence the compromise an operator may be compelled to take in practice, but it should certainly not enter into consideration of what is rational treatment of a sick mouth. Particularly should that be true of scientific papers and clinics before the leading authorities of any specialty. It seems to me that we should first find out what is required to bring about a successful result, and then search for the ways and means. I believe I have tried all the so-called simpler ways, but personally

have never been able to make them produce even a comparable result. Believe me, I wish far more than you that some one could find a simple way. I would welcome it with open arms. But I do insist that it will create conditions which will make it possible for nature to come to our aid, not merely make it possible for vigorous physical therapy to control the gross pathology while the destructive process continues.

I trust this has not been too lengthy a discussion, but to say less could leave a very false impression. Eccentric relation is merely a part of the functional syndrome. It cannot be treated as some isolated entity. Indeed, I have seen cases in which by grinding an eccentric relation was corrected without considering the rest of the picture, and it actually exacerbated the condition, making it more acute. Not one case, but many. At best, grinding alone can only make a chronic process out of an acute one. At worst, it can make matters much worse. The other alternative you suggest, a combination of grinding, rebuilding, and orthodontia is actually less practical than what we are doing.

F. RAYMOND GARVEY, D.D.S.
Saint Paul, Minn.

I appreciate the opportunity to discuss your Initial Statement because I am quite familiar with the work Dr. Granger is doing in mouth rehabilitation, and most particularly, because at present in my practice we are applying the basic knowledge of articulation established by the Gnathological Society. We are also using the equipment of their design as a necessary part of the practical application of this knowledge in the reconstruction of the mouths of many individuals. Without this type of service, these particular individuals are earmarked to lose their natural teeth because of extremely advanced periodontal involvement.

Analysis of the Initial Statement indicates to me that you have failed to grasp the essence, or the complete meaning, of Dr. Granger's paper. Within its pages can be found, as far as I know, the first logical explanation for, and the first logical approach to, a solution of many perplexing problems presented by the extremely complex picture of periodontal disease. Space does not permit a review of these problems but I am sure they are familiar to all of us.

An exhaustive review of the literature relative to the etiology of periodontal disease leaves the reader bewildered because of the conflicting opinions of various investigators. The etiologic factors most common in the literature can be divided into two great classes; the intrinsic, or systemic factors, and the local, or extrinsic factors. Obviously, they both enter into the picture, but there exists a gap between these two groups of factors which is not explained in the literature. Dr. Granger's treatise on the role of function and its relation to the metabolic requirements of the tissues, bridges that gap; and thereby brings the entire picture into much sharper focus and explains many of the apparent mysteries of the problem.

You imply that the "elaborate" and "expensive" system of developing an ideal articulation is not necessary in the vast majority of cases. Whether that statement is correct or incorrect depends on the type of cases to which you refer. The degree of involvement in any particular case will determine what treatment is required to restore that mouth to health and function.

You also imply that so-called "simpler" and "cheaper" methods of restoring normal function will produce the same results. With that, I cannot agree. I will admit, however, that one may produce a result which is good enough for a given individual by haphazard methods of articulation. This is possible because that patient's metabolic level does not require an ideal articulation.

Certainly the majority of cases referred to a periodontist for treatment could be classed as advanced cases of periodontal involvement. Why are they advanced cases? They can be considered advanced because a goodly portion of the supporting bone has disappeared, which means that the functional demands on the remaining bone will be in

excess of what that bony support can tolerate. It is those cases which fall into this category where it becomes necessary to reduce the functional demands on that bony support to an absolute minimum. That can only be done by the establishment of a most ideal articulation.

I have for almost twenty years, working with the cooperation of an extremely capable periodontist, using several different methods of reconstruction, tried desperately to save what remains of natural dentitions ravaged by periodontal disease. Some advanced cases have been very successful and other have failed. Many of these cases presented with various types of restorations placed with no regard for proper function, and instead of serving as a treatment for sick teeth, actually hastened the progress of periodontal disease.

A review of these cases clearly indicates that the nearer we approached an ideal articulation, by whatever method used, the more successful the case. However, I am certain that the time and effort we expended in building these cases far exceeds the time and effort expended when cases are constructed with the aid of Gnathological instruments, and I must add that the results attained by the so-called simpler methods, were inferior to those now attained.

No doubt a certain percentage of the population does not care whether they save their teeth or not. But let us here concern ourselves with that large percentage who have the proper attitude and definitely do want to save their teeth, and will make great sacrifices to do so, regardless of their financial status. What shall determine the nature and extent of our remedies for these individuals? It certainly should depend on first, our ability to recognize what the patient requires, and secondly, our ability to fulfill those requirements; but most certainly, should never be determined by the size of the patient's bank account.

It seems logical and obvious to me, that if we expect to restore mouths to normal function, we must first have a basic understanding of exactly how the human mandible functions. That knowledge is presently available, and has been for more than ten years, through the tireless efforts of the Gnathological Society of California. This knowledge is so basic and indisputable that the profession, and particularly periodontists, dare not ignore it. Unfortunately, mandibular movements are extremely complicated and their duplication, at the present time, naturally requires the use of instruments which appear complicated and expensive. This fact undoubtedly explains the widespread non-acceptance of this vitally important contribution to dental science. However, it is reasonable to suppose that if adequate effort and funds are directed to further studies of jaw motion and the functional relations of the teeth, much simplification of present procedures will result. If the medical profession had adopted the same attitude toward the discovery of Penicillin as we have toward Gnathology, it would still be available only to the privileged few.

The Gnathograph and Gnathoscope should not be considered as just another set of gadgets which are too complex and too expensive for general use; but rather, as the only instruments which at present will accurately record and reproduce jaw motion. They are merely a means to an end—our constant aim—a better basic understanding of oral physiology. If every member of this Academy would take the time to study, "A Research Report" by McCollum and Stuart, which appeared monthly in the *Dental Items of Interest* from June, 1939, through February, 1940, the knowledge thus gained would serve as a common ground upon which to make a fair appraisal of Gnathology.

J. RICHARD LOUGHRY, D.D.S.
Cleveland 15, Ohio

Many of the modern teachings of ideal occlusion and the correction of malocclusions are becoming so complex, with the advocation of the use of articulators, face-

bows and other mechanical means of trying to obtain a satisfactory result, that the basic concept of occlusion and its function is lost.

This basic concept, to me, is that we are dealing with human beings with many or most of their natural teeth present, each of these patients has a different occlusal relationship, which may be to that particular patient, a natural occlusion. The differences which occur in various individuals may be due to a host of factors as noted in the "In Our Opinion" Department of the 1946 Journal (17:80-91 April, 1946).

It is impossible, in my opinion, to reproduce the majority of occlusal movements in a particular individual on a mechanical articulator where that individual has a major portion of his natural teeth still present. The most satisfactory results are obtained by studying the intercuspal relationship of the teeth, in the mouth and traumatic interference in anterior, and lateral movements of the mandible, after adjusting undue stresses of any teeth in centric relation. Occlusal balancing by grinding, meticulously done, in the great majority of cases will correct these undue stresses. Extreme, Class II malocclusions or narrow arches with crowding of the teeth may require artificial opening with splints or orthodontic treatment.

Occlusal adjustment in the treatments of periodontal cases, and of those cases, which as yet, do not show signs of periodontal disease, is one of the most important factors in successful treatment and preventions.

It is not a simple procedure, it is one which is often overlooked, and it is one most successfully carried out by studying each individual human dentition in operation without the adjunct of artificial reproductions which do not truly reproduce the natural alignment and movement of the teeth.

B. B. MCCOLLUM, D.D.S.
Los Angeles, Calif.

In the first place, I agree in general with the six factors that you have given as the cause of periodontal disease, but I think you have used the word "occlusion" incorrectly. It is the articulation which I think causes the interference and not the mere closure of the mouth. I will discuss in particular the practice of grinding teeth directly in the mouth in order to bring about proper articulation.

In my opinion, this manner of attempting to correct articulation is mechanically impossible. There is not enough material in the teeth to allow for bringing about the desired result. Temporary relief can be obtained by grinding off certain cusps or planes of teeth that have been injured by malarticulation, but no results can be obtained that could be called permanent—a year or two is the ultimate. Other planes or cusps of the teeth are merely brought into a position to bear the stress and the patient is deceived by the relief obtained. However, with the teeth unsupported by the proper articulation, as they would be in that case, the patient in due time finds himself in a worse condition than he was before the operation was started.

I have witnessed many attempts at grinding and have seen the results where teeth have been ground three or four times in five years. The patient obtained relief for a year or more after the first grinding, but after each re-grinding the results only lasted a few months, and in some cases the patient was miserable until he had the articulative surfaces of the teeth restored.

In my opinion it is just as impossible to accomplish the articulation of natural teeth by grinding as it is to align the bearings on a machine such as an automobile by attempting to grind the defects of wear out of them. We all know that it requires a mechanical alignment of such bearings to bring them into usefulness. In other words, they have to be built up to a proper condition. The original automobile was not made with the perfection we know today, and we are all conscious of the fact that the old-time automobiles had to be worn in which was merely a process of wearing them out. If they are right in the first place, we all know that well aligned bearings last a long time.

I have seen clinics on how to grind teeth into articulation, in fact a number of them,

and I want no better proof than the results shown on models at these clinics to prove that it could not be done. In all the clinics that I have seen they merely brought new planes into apposition and did not accomplish the needed articulation in any sense of the word.

Doctor MacQueen gave a clinic at Houston before the Academy of Restorative Dentistry in 1938, I believe, showing the impossibility of so-called occlusal grinding. It was one of the most graphic demonstrations I have ever seen.

ROWE SMITH, D.D.S.
Texarkana, Arkansas-Texas

After studying the initial statement which you enclosed I find myself in almost complete agreement with it, especially since you are including the principles laid down by McLean (id. 1939), Schuyler (J.A.D.A. 22 1193-1202 July 1935), S. C. Miller et al (Textbook of Periodontia 1944), H. J. Leonard (id. 1943), and others. In addition, I would specifically like to include "Correction of Occlusal Disharmony of the Natural Dentition," Clyde H. Schuyler, D.D.S., The New York State Dental Journal, Vol. 13, No. 8, pages 445-462, October 1947.

Special emphasis should be given the statement on page 2, paragraph 3, of your initial statement—"providing the tissues are kept healthy from a standpoint of sanitation, nutrition, and blood supply." My experience has proven that adequate nutrition will improve the integrity of the dental supporting tissues, thereby enabling them to withstand increased and unnatural occlusal forces without serious disturbances or damage. Any disturbance or interference with proper utilization of adequate nutrient intake should be corrected wherever possible.

I am also in agreement that it is obvious that a small percentage of people who are suffering from extensive damage of their supporting tissues due to periodontal disturbances fail to make an acceptable response to all types of periodontic treatment, including nutritional and appropriate medical therapy. Undoubtedly, these cases will be benefited by the occlusal reconstruction treatment as advocated by Drs. Granger, McCollum and others, provided sufficient dental supporting bone remains, if they are able to meet the extraordinary financial requirements.

I am convinced that where there is clinical and roentgenologic evidence of malrelationship of the teeth, study casts should be properly mounted on an adjustable instrument, in order to afford the most accurate diagnosis possible and also to ascertain the potential result to be achieved by corrective grinding, reconstruction, etc.

I would like to mention another traumatic condition which predisposes to periodontal disease: that is, the unfavorable wedging effect of a lower dental arc which is too large for the opposing upper dental arc, as a result of buckling ("Periodontal Conditions Associated with Buckling of the Lower Anterior Teeth: Etiology, Diagnosis and Treatment," published in Journal A.D.A., Vol. 34, pages 303-309, March 1, 1947; especially p. 306, figure 5). Owing to the displacement of the lower anterior teeth, excessive traumatic force is produced on the supporting tissues of one or more of the upper or lower anterior teeth, which results in pocket formation, drifting, and formation of diastemas. This disturbance can be permanently corrected only by adequate reduction of the displacing pressure caused by the occurrence of buckling. (I had an opportunity to show this to Dr. Clyde Schuyler last year when I was attending a week's seminar on occlusal and temporomandibular joint disturbances, which was conducted by him and Drs. John R. Thompson, Harry Sicher, Balint Orban, and Bertram Downs in Colorado Springs, under the auspices of the Colorado Foundation for Dental Research. Dr. Schuyler told me he thought it was worthy of more consideration, and thanked me for bringing it to his attention.)

You are to be complimented upon the very complete and unbiased manner in which you have prepared this initial statement, and I believe that it will help to clarify much of the misunderstanding and misinformation

about this aspect of periodontal disease. It is timely and should be of great interest to periodontists and nearly all dentists.

SIDNEY SORRIN, D.D.S.
New York 22, N. Y.

It is a pleasure to discuss this phase of periodontia which is rapidly being recognized by the entire profession. I am quite in agreement with the opinions which you have expressed in your splendid review of the facts presented. In the study of periodontal diseases, traumatic occlusion is only one of the many factors which may be responsible for the breakdown of the supporting tissues. This one factor however, is so closely related to mouth rehabilitation that dentists are endeavoring to reach a better understanding of it and how occlusal equilibration may be satisfactorily accomplished.

By occlusal equilibration is meant an arrangement of the teeth whereby stresses on all the teeth are innocuously distributed in any position of the jaw. By establishing this condition, prematurities are eliminated and simultaneous contact of as many teeth as possible in the various positions of the mandible is secured. We must think not only in terms of centric relation and centric occlusion, but also of protrusive position, protrusive excursion, lateral excursions of the jaw, and free way space. In these movements and positions of the jaw, condylar guidance, incisal guidance, musculature, wear of teeth and tissue resistance play a definite role in determining procedures for correction.

Each case must be determined on its individual merits, and occlusion varies with age, due to stress and wear of the teeth. Recently* Dean Walter H. Wright of New York University made the statement in speaking of occlusion. . . . "We as a profession are now concerned with the *individual* and his *function* while *averages* and *anatomic ideals* are reserved for theoretical discussion. We now regard the individual with his peculiar dentition and related function as the criterion of dental treatment. . . . Each individual differs from another. It is therefore obligatory that we recognize these variations and accommodate them in order to provide functional, comfortable and health promoting dental treatment."

This is important in any condition where occlusion contributes greatly to the breakdown of the periodontal structures. From the definitions of centric relation and centric occlusion, it is not necessary for these positions to coincide. In adults they are rarely in the same position and yet may be in harmony with no periodontal pathosis or temporomandibular discomfort. At times a severe discrepancy may occur and by the grinding of prematurities, periodontal pathosis and temporomandibular discomfort may be relieved.

For many years, we have been able to teach fundamentals of occlusal equilibration to our undergraduate and postgraduate students alike. It is difficult in a discussion of this nature, to go into the many details. However, an outline of procedure may be helpful. After casts, charting roentgenographic examination has been made, we examine the various movements of the jaw and note the following:

1. Whether this is a case requiring complete mouth reconstruction because of loss of vertical dimension or other factors.

2. Whether orthodontic interference would best be advised.

3. Whether selective grinding can accomplish the desired result.

4. Whether a combination of these factors would be the best treatment planning.

If grinding is the procedure of choice, the following is considered:

1. Preliminary Grinding: This is a procedure whereby, food impactions are relieved, facets and premature wear are reduced, and esthetics and patient's biting habits are studied. We must be careful in all grinding processes, that we do not interfere with esthetics, or phonetics or induce sensi-

*Part of a Symposium on Occlusion in Dentistry for Dean's Day, Columbia University, February 17, 1950.

tiveness. We must remember that we are concerned with natural teeth and are limited with the amount of grinding which can be accomplished.

2. Our next steps in order are centric occlusion, protrusive position and excursion and lateral excursions of the jaw. Time does not allow for detailed discussion, but several points can be considered at this time which are pertinent to our opinion.

When we grind for any position, we must always check to see whether we may be disturbing an equally important contact in another position of the jaw. Thus objectives in all positions of the mandible must be clearly understood.

Most of the discussion seems to be related to centric relation and centric occlusion. In natural teeth, a centric occlusion is present which may or may not be in harmony with centric relation. By means of selective grinding, using pink wax, articulating paper, patient's responses to questions as to where teeth seem to strike, visual study, roentgenographic examination, lesions on soft tissues and sound on closure of the jaws, as guides, we reduce premature contacts which prevent correct closure in this position. Often we may be forced to disocclude a tooth, which requires physiologic rest. In another instance we are forced to take a tooth out of contact in this position and depend upon its contact in another excursion to prevent extrusion. We must consider the age of the patient and the position of the jaw at that age which may be relatively normal and the resistance which has been offered by the tissues. Tolerance of forces to maintain equilibrium between the applied force and resistance forces are observed in many of our cases. Often the destruction about the teeth is due to a combination of forces in all movements of the jaw, rather than to forces applied in only one movement or position of the jaw. That is why occlusal equilibration in all movements of the jaw should be the ideal.

Musculature and temporomandibular joint and tissue resistance are important factors in guiding our procedures which should be tempered with common sense and not dogmatic hard rules. Nature in dentistry or medicine is a great aid in determining our procedures and helping us in many of our problems. It should be given every opportunity.

Many cases of completed mouth rehabilitation have caused serious temporomandibular joint discomfort, and loss of teeth, because Nature often rebels against the strained and unnatural changes. Judgment and complete knowledge of fundamental principles of occlusion must be uppermost in the minds of those who are recommending mouth rehabilitation which includes changes in vertical height and new guiding inclines in the other movements of the mandible.

In many cases it is the only solution to the problem. In other cases, orthodontia may be helpful. However, whether occlusal equilibration is accomplished by restorations, orthodontia, grinding or combination of these, fundamental principles in gaining occlusal harmony in all movements of the teeth is essential.

WILLIAM A. THEMANN, D.D.S.
Newark, N. J.

It is a privilege to be able to comment on the continued study of the occlusal factor. Ernest Granger's emphasis on the fact that centric relation and centric occlusion may not coincide is an extremely well overlooked fact by a high majority of dentists. It should, however, be one of the most important considerations in any restoration. The piece of dentistry placed in the patient's mouth to correct one defect may itself be the means of creating disharmony in centric relation or in one or more of the eccentric movements of the mandible.

H. J. Leonard's states, "We should keep dentistry as simple and inexpensive as possible and still accomplish our purpose of maintaining the teeth in maximum health, function and beauty." I agree with him most wholeheartedly. An elaborate articulator like B. B. McCollum's is unnecessary in my hands

to find either a premature point of contact or to establish proper articulation for rehabilitation. Existing articulators and technics do not accurately reproduce all mandibular movements, making final adjustments necessary in the mouth. A simpler articulator like Hanau's for restorations and occlusal adjustments with meticulous balancing in the mouth using nature's own articulator and the jiggling of the mandible in a relaxed position seems more accurate and less time consuming. After determining that there is a premature point of contact with the jiggling technic, the patient can guide you by informing you of the approximate area of contact.

Various materials have been used to cover the occlusal surfaces of the teeth in the suspected area. A thin sheet of black carding wax is more resistant than pink base plate wax. A thin easy flowing material like melted black carding wax or especially Kerr's pink disclosing wax painted or flowed on the dried tooth surface makes the jiggling technic of punching a hole in the wax simple and quick. The contrasting color of the tooth surface showing through the wax permits marking the premature contact with a pencil. This method can also be used to show the pathways of the cusps as they glide along the opposing planes.

The technics for occlusal adjustment previously described by Sorrin, Schuyler, Leonard et al, are used for balancing the case and reshaping teeth to lessen and redirect horizontal forces to convert them to tolerable inter-alveolar forces. Many cases can be completely rehabilitated by using restorations including fillings, bridges or partials in local areas where required and reshaping and balancing the remaining teeth to conform to the basic occlusal adjustment principles. A mobile cuspid, loose anterior teeth etc., that do not respond to balancing of the occlusion, may be splinted to firm teeth. The remaining teeth may satisfy requirements with proper occlusal adjustment.

In some cases complete rehabilitation of all the remaining teeth in the mouth is the only solution. Where finances will permit, this method is the one of choice. However, something less ideal can usually be designed or full dentures may be the unfortunate solution in cases of limited finances. Complete rehabilitation is a marvelous means of balancing forces and should be used when the patient can be convinced of its necessity and has the means available.

The relief of premature points of contact takes its place as a necessary procedure with other imbalances in centric and eccentric causing periodontal traumatism. A large percentage of our patients have premature contacts; some cause disturbances, other do not. Many cases of various types of distress of the temporomandibular joint and the muscles of mastication have been completely helped by removing these premature points of contact. The technics short of rehabilitation and complete rehabilitation should be used each in its place to bring the greatest degree of health and happiness to our patients.

Concluding Statement

HAROLD J. LEONARD

I greatly appreciate the kind manner in which the discussors have written especially Garvey and Granger whose methods were criticized in the Initial Statement. I am glad this subject has been presented for it brings out the general ideas of periodontist leaders in this field and also those of the Gnathological Society of which Granger, Garvey and McCollum are members. Certainly the Gnathological Society ideas are more clearly presented here than they have ever been previously in the periodontal literature to my knowledge.

Friedlander's comment that the expense of treatment should not enter a discussion of its scientific merits is well taken. We should seek after and determine what is best for the individual and Society and if that proves impracticable for any reason we can always compromise to obtain the best possible under the circumstances.

I think perhaps the main difference in philosophy of treatment between most periodontists who are intensely interested in occlusion as a factor in periodontal disease, and members of the Gnathological Society is due to their approach to dentistry and their background experience. The periodontist has tissue health as the central point of his thinking. He is concerned with changes due to irritants, to tissue tone, metabolism, blood circulation, nutrition, and tissue chemistry. To him occlusal stresses which aggravate or initiate tissue changes leading to periodontal disease or breakdown are just one type of irritant which must be eliminated by any means available. He sees so many healthy mouths with cusps hitting end to end and with many other faults in occlusion that he does not look on ideal occlusion as necessary. Such defects may impair masticatory function somewhat but if they do not create lateral thrusts on the teeth, they seem to be well tolerated.

Members of the Gnathological Society on the other hand are primarily restorative dentists to whom the mechanics of occlusion is the central point of thought. Not being highly trained in periodontia they have not succeeded in getting advanced periodontal cases back into health by means used by well trained periodontists and have instead devoted their effort to reducing occlusal irritants to zero.

Whether or not there are cases in which complete occlusal reconstruction to obtain ideal intercuspation in relation to centric relation, is necessary, I am not prepared to say. In a fairly wide experience I have not seen cases which I think failed because this was not done. Some fail but for other reasons. However, I may be wrong in this. The Gnathological Society through Dr. Granger has introduced a concept important to periodontists and which should be further explored. Whether or not the group is on the right track will be determined by experience of cooperation between members of the group and well trained periodontists competent to evaluate the results obtained. There have been so many disillusioning experiences with enthusiastic groups who thought they had ideal mechanical articulators and who reconstructed occlusions according to preconceived mechanical principles, with disastrous results, that an experienced periodontist is inclined to be pretty reserved in accepting a method which is as difficult as total occlusal reconstruction using the McCollum technics.

Conference on Periodontal Disease

A conference on periodontal disease will be jointly sponsored by the Dental Study Section of the National Institutes of Health, Public Health Service, Federal Security Agency, and the United States Army, United States Navy, United States Air Force and Veterans Administration.

This conference will be held at the Institute of Pathology, Western Reserve University, Cleveland, Ohio, on May 19th.

The program will include the following participants. Ample opportunity will be provided for discussion.

Dr. Isaac Schour, Terminology and Classification.

Dr. Maxwell Karshan, Etiology.

Dr. Harold Hodge, Calculous Formation.

Dr. Donald Kerr, Systemic Relations.

Dr. Henry Goldman, Pathological Changes in Soft Tissues.

Lt. Col. Joseph Bernier, Pathological Changes in Hard Tissues.

Dr. Clifton Dummet, Orthodontia and Periodontal Disease.

Dr. Irving Glickman, Tissue Metabolism.

Dr. David Marshall-Day, Epidemiology.

This conference will be open to all who are interested in periodontal disease.

For further information or hotel reservations write to Thomas J. Hill, Western Reserve University, 2085 Adelbert Road, Cleveland 6, Ohio.

Announcements

Baylor University College of Dentistry is giving a two-day post graduate course in Periodontia for general practitioners who have not had recent work in Periodontia, to be held in the Periodontia Clinic of the New Dental Building, Friday, April 21 and Saturday, April 22, immediately before the Texas state meeting. The course will be under the direction of Samuel R. Parks, D.D.S., F.A.C.D., professor and chairman of the department of periodontia in Baylor University, who will be assisted by members of related departments.

The Journal of Periodontology has entered into an agreement with University Microfilms, Ann Arbor, Mich., to make available to libraries issues of a number of McGraw-Hill publications in microfilm form.

One of the most pressing problems facing all types of libraries today is that of providing adequate space for a constant flood of publications. Periodicals pose an especially difficult problem because of their bulk and number.

Microfilm makes it possible to produce and distribute copies of periodical literature on the basis of the entire volume in a single roll, in editions of 30 or more, at a cost approximately equal to the cost of binding the same material in a conventional library binding.

Under the plan, the library keeps the printed issues unbound and circulates them in that form for from two to three years, which corresponds to the period of greatest use. When the paper copies begin to wear out or are not called for frequently, they are disposed of and the microfilm is substituted.

Sales are restricted to those subscribing to the paper edition, and the film copy is only distributed at the end of the volume year.

The microfilm is in the form of positive microfilm, and is furnished on metal reels, suitably labeled. Inquiries concerning purchase should be directed to University Microfilms, 313 N. First Street, Ann Arbor, Michigan.

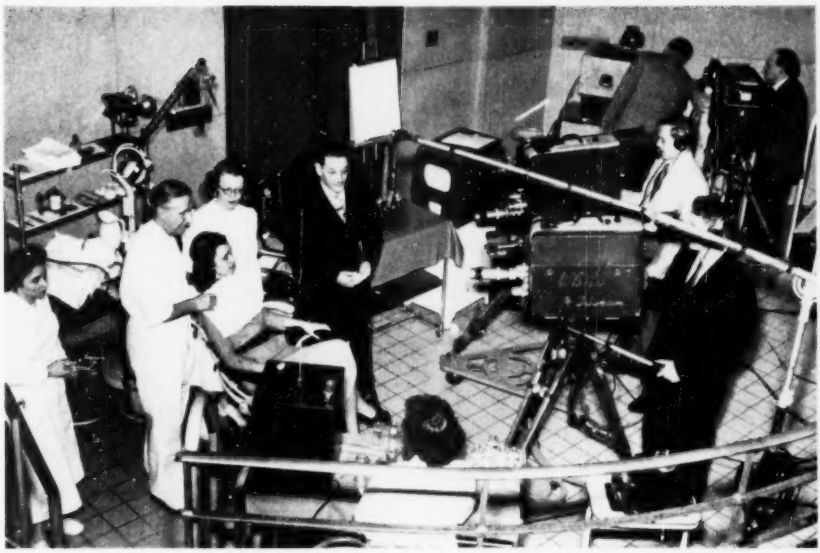
Bernhard Gottlieb, M.D., D.M.D., L.L.D.

March 16, 1950

Dr. Gottlieb died suddenly in

Dallas, Texas, on March 16, 1950.

Details will be published in the next issue.



Periodontia on Television

One of the features of the two day Post-Graduate course given in February at the University of Illinois College of Dentistry in Chicago was the use of television. Dr. Balint Orban demonstrated technics, and results of treatments on patients with periodontitis before the camera, and the "students" were grouped around several receiving sets a few floors above.

The technical aspects of the procedure were interesting. Three cameras were used, two focused on the patient at different angles, and one set up for projections of microscopic specimens. One receiving set was placed so that the operator could see what was being televised. The results were good, in general, and allowed a large group to see the clinical aspects of the problem under discussion, then the radiographs of the patient, and finally histological sections of comparable cases.

The picture above showing Dean A. G. Brodie introducing Dr. Orban, was sent to the Journal by Dr. Orban.

Seminar on Oral Diagnosis Colorado Dental Foundation

The Colorado Dental Foundation announces its Fourth Annual Seminar to be held June 18-22, 1950, in Colorado Springs, Fine Arts Center. Oral Diagnosis is to be the theme of the meeting.

The program has been designed to give special emphasis to the differential diagnosis of oral diseases and to the diagnosis, ill effects, and treatment of occlusal disharmonies.

Essayists who will lead the discussions include:

Dr. Kurt A. Thoma, Professor of Oral Surgery and Oral Pathology, School of Dentistry, Harvard University.

Dr. Charles A. Stuart, Research Worker in Articulation of Human Teeth, Ventura, California.

Dr. Joseph P. Weinman, Professor of Oral Pathology, School of Dentistry, University of Illinois.

Dr. Harry Sicher, Professor of Anatomy, School of Dentistry, Loyola University, Chicago.

Dr. John R. Thompson, Professor of Orthodontia, School of Dentistry, Northwestern University, Chicago.

Dr. Balint Orban, Director of Research, Colorado Dental Foundation, Colorado Springs, Colorado.

For further information write the Colorado Dental Foundation, 629 North Nevada Avenue, Colorado Springs, Colorado.

Academy of Dental Medicine Meets June 24-25, 1950

The fourth annual meeting of the American Academy of Dental Medicine will be held at the Copley Plaza Hotel, Boston, Mass., June 24-25, it has been announced by Dr. William M. Greenhut, New York, National Secretary.

Dr. George F. Clarke, Boston, Chairman of the Annual Meeting Committee, discloses that because rapidly expanding biological research will have a pronounced effect on the treatment and care of the oral cavity, the two-day program has been arranged to take advantage of Boston's excellent research facilities.

The meeting program is as follows:

Saturday, June 24.

10:00 Business Meeting

12:30 Luncheon. Presentation of Honorary Membership to Dr. Philip E. Adams, President of the American Dental Association.

2:00 Enzymology and Biochemistry of the Mouth by Dr. Irving W. Sizer, Associate Professor of Physiochemistry, M.I.T.

2:45 Electromicroscopy by Dr. Cecil E. Hall, Associate Professor, Biophysics, M.I.T.

3:30 Use of Isotopes in Biological Research by Dr. Arthur K. Solomon, Associate Professor, Physiochemistry, Harvard.

Sunday, June 25

10:00 Business Meeting

12:30 Luncheon

2:00 Symposium on Oral Cancer

Dr. Charles C. Lund, Moderator, President, Mass. Section American Cancer Society

Dr. Ira M. Nathanson. Pathology, physiology, and biology of Cancer.

Dr. David Weisberger. Cancer diagnosis, differential diagnosis, pre-cancerous, and non-malignancies.

Dr. Thomas Anglen. Prevention and treatment of Cancer, including Surgery and X-rays.

Registration covering both days, including luncheons and admission to scientific sessions, is ten dollars. Applications and requests for hotel reservations should be sent to Dr. George F. Clarke, at 10 Postoffice Square, Boston, Mass.

We Apologize!

The Editor of the Journal of Periodontology finds it necessary to apologize to the readers, and issue a plea for help. Because of the success of the subscription campaign organized by Dr. E. Romle Romine, all of the extra copies of the January 1950 issue of the Journal have been distributed. This means that in the future new libraries will be unable to get complete files. If, therefore, any readers are finished with their copies they can perform a real service by returning them to the Editor. Sufficient copies of the current issue have been ordered, so that the subscription drive need not slow down!